ACTA PÆDIATRICA

Vol. XXXIV. Supplementum I.

1947

Almqvist & Wiksells Boktryckeri-Aktiebolag

UPPSALA 1947

ACTA PÆDIATRICA

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30 POLHEMSGATAN, STOCKHOLM

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ACTA PÆDIATRICA

FROM THE PEDIATRIC CLINIC OF KAROLINSKA INSTITUTET AT
KRONPRINSESSAN LOVISA'S CHILDREN'S HOSPITAL, STOCKHOLM.
HEAD: PROFESSOR A. LICHTENSTEIN, M. D.

HEART ARRHYTHMIAS IN CHILDREN

BY

BERNHARD LANDTMAN

ACTA PAEDIATRICA, VOL. XXXIV. SUPPLEMENTUM I

HELSINGFORS 1947

HELSINGFORS 1947
MERCATORS TRYCKERI

TO

PROFESSOR ADOLF LICHTENSTEIN

in gratitude and admiration

Contents

PREFACE

The present work has been carried out at the Pediatric Clinic of Crown Princess Lovisa's Children's Hospital in Stockholm. The investigations, which began in September 1945, are part of a comprehensive program of work at the Heart Station of the Hospital.

To the Head of the Hospital, Professor ADOLF LICHTENSTEIN, I wish to express my most heartfelt thanks for his great kindness in according me the privilege of carrying out these investigations, and also for the support and encouraging interest which he has shown me all through. At the same time I am deeply grateful for the teaching which I had the advantage to enjoy from him in the Clinic beside my research work.

To my teacher, the Chief of the Heart Station, EDGAR MANNHEIMER, M.D., my particular thanks are due for the invaluable help and the enriching impulses continually received from him in doing my work. Also for the knowledge obtained during the daily activity of the Heart Station under his stimulating guidance I express my cordial thanks.

The work has been made possible by a grant from Nordisk Insulinfond for which I am most grateful.

Since returning to Finland in March 1946 I have received financial support for the completion of the work from the Chancellor of Helsingfors University and here express my gratitude to him.

The translation has been done at the expense of the British Council in Helsingfors and I much appreciate their help and interest. Finally I thank Miss AGNES DAWSON B.Sc. (Econ.) for translating my work.

Helsingfors, December 1946.

Bernhard Landtman.

INTRODUCTION

The heart arrhythmias were formerly regarded as almost exclusively affecting adults. Thus, in studying these disturbances attention was chiefly paid to this age-group. Our knowledge of arrhythmias in children, on the other hand, is still very incomplete. Considering the large number of detailed monographs on these disturbances in adults which have been published ever since the days of WENCKEBACH and WINTERBERG, it is curious that no great work has been written on its occurrence in children. One of the reasons for this is that modern methods of examination have only rather lately received adequate attention in pediatrics. However, heart arrhythmias can present all their known forms in childhood and there they certainly do not play the subordinate role formerly attributed to them.

In judging this problem in children one meets conditions, due to the special pathology of childhood, which are widely different from those found in adults. Thus, such diseases as coronary sclerosis and hypertension, which in older people are the most usual causes of disturbances in the heart action, are almost unknown in children. For this reason arrhythmias in children can very rarely be judged on the basis of experiences obtained from adult material. From a pathogenetic point of view, moreover, it is of the greatest importance to have as complete a knowledge as possible of the disturbances in the heart action during an early period of life. Also as regards the prophylaxis of heart diseases this is important, as different forms of arrhythmias arising in childhood can remain or recur later in life. Against this background arrhythmias in children seem to be a subject of great interest.

The object of this investigation has been to obtain an insight into the problem of arrhythmias in children on the basis of observations made of a large amount of material. Attention has here been directed to the study partly of the frequency of different types of these disturbances, partly of their clinical manifestations.

As an introduction sinus arrhythmia was studied under normal and pathological conditions. As regards the mechanism of the origin of arrhythmias the continuation of the work is divided into two main parts. In the first part the disturbances of stimulus formation are treated, in the second the arrhythmias due to conduction disturbances. Among the former sinus tachycardia and sinus bradycardia are not treated, as they are outside the domain of arrhythmias in their real significance. Sinus extrasystoles and sino-auricular block, which may possibly be included in the sinus arrhythmias have, because of the mechanism of their origin, been treated in the chapters entitled »Extrasystoles» and »Block» respectively.

Finally, the extent to which premature infants show heart arrhythmia to a special degree has been discussed in a separate chapter.

Material and method

At the Heart Station in Crown Princess Lovisa's Children's Hospital 5,600 children under the age of 16 were examined during the years 1936—1946. At different times a total of 8,650 records have been taken from these children and they have all been studied in collecting the arrhythmia material used here. Pathological forms of arrhythmia were found in 126 children (2.25 per cent). Included in the material were also 200 healthy children to determine the physiological sinus arrhythmia.

The material at the Heart Station is composed partly of the patients treated in the hospital and partly of the heart cases investigated polyclinically. During recent years this latter group has tended to increase continuously. Thus since 1938 all children with suspected heart disease were sent from all the elementary state schools in Stockholm to the Heart Station for examination.

Electrocardiographic examination. Since 1938 Elmquist's three-lead-electrocardiograph (Triplex) has been used at the Heart Station. Before that the apparatus used was a one-lead-electrocardiograph of ordinary construction. Generally the patients were examined in a lying position. The electrodes were laid with clips round the upper arms and round the left lower leg. Between the clips and the skin was laid a compress damped with a solution of calcium carbonate. For little children similar but smaller clips were used. The length of the films at the examination corresponded as a rule to a period of 20—30 seconds.

Phonocardiographic examination. The method worked out by MANNHEIMER for calibrated phonocardiography has been in general use at the Heart Station since 1938. This method makes

possible an objective registration of the heart's sound phenomena. Its principle is shortly as follows:

Through a microphone, which transforms the sound phenomena into electric impulses, the differences in the voltage arising in this way are led to the phonocardiograph. In this the electric impulses are passed through an amplifier into a number of canals, each of which is equipped with its special amplifier, and thence to a system of filters which divide the sound phenomena into different frequency ranges. After having passed through an amplifier the impulses are finally led to the oscillators of the electrocardiograph which simultaneously with the electrocardiogram record the phonocardiogram on a passing film. In this way it is possible to judge the position of the heart sounds and murmurs at the different phases of the pulse period.

As the impulses within the different frequency ranges are in this way registered separately, six phonocardiograms are obtained, and these form, so to say, part pictures of the whole range in which the heart sounds and murmurs, even such as the ear does not apprehend, occur. In the lowest frequency range, below 100 cycles per second, lie the lowest heart sounds. In the other five are registered both the murmurs and the overtones of the heart sounds. These six frequency ranges lie between 50—175, 100—250, 175—400, 250—500 and 500—1000 cycles per second respectively.

Besides the arrangements for amplifying and filtering there is an apparatus for the calibration of the sounds' amplitude. It consists of tube oscillators which give out tones of known frequency and amplitude with which the voltage of the registered impulses, reckoned in millivolts, can be compared. The microphone is calibrated in a special apparatus so that the vibrations can be expressed absolutely (dyne/cm^2). In this way it is possible to reproduce the method.

Statistical calculation.

Current statistical formulae have been used in judging some of the results.

As the number of observations, n, in the different series does not exceed 50, the standard deviation, δ, has been calculated according to the following formula:

$$\delta = \pm \sqrt{\frac{\Sigma a^2}{n-1} - M^2}$$

where a indicates the different observations and M their mathematical mean.

When n exceeds 50 the following formula has been used:

$$\delta = \pm \sqrt{\frac{\Sigma a^2}{n} - M^2}$$

The standard error of the mean, ε (M), has been obtained from the formula:

$$\varepsilon (M) = \pm \frac{\delta}{\sqrt{n}}$$

The standard error of the difference, ε (D), has been calculated from the formula:

$$\varepsilon (D) = \pm \sqrt{m_1^2 + m_2^2}$$

where m_1 and m_2 indicate the standard errors of the means in question.

Explanation of the figures.

The electrocardiograms (ECG) and phonocardiograms (PCG) are reproduced in half scale. Time scale division is 1/50 second in all figures except in fig. 14 and 15 where it is 1/20 second.

Sinus arrhythmia

The heart of a healthy individual does not beat quite regularly. The sinus node, the normal pacemaker of the heart, is under the influence of the extracardiac nerves, partially accellerating, partially impeding. The general view is that sinus arrhythmia chiefly arises in consequence of the variations in tonus in these nerves, of which the vagus plays a dominant part. By eliminating the effect of the vagus, e.g. by the use of atropin, a decrease of the sinus arrhythmia can be brought about (FLEISCH and BECKMANN 1932).

Sinus arrhythmia specially characterises childhood and is therefore often called juvenile arrhythmia (MACKENZIE). The so-called respiratory arrhythmia connected with the breathing is the dominant one, and is characterised by an increased heart frequency at inspiration and a decreased one at expiration. FRIBERGER (1912) states that this form of arrhythmia occurs in 100 per cent of healthy children. SHOOKHOFF and TARAN (1931), also MENGOLI (1936) give corresponding figures of 73 and 87 per cent respectively LYON (1945), in speaking of sinus arrhythmia in children, points out that it is chiefly conditioned by the respiration.

Opinions are still divided as regards the respiratory arrhythmia's more immediate origin mechanism. According to HERING's so-called reflex theory (1871), which has lately been accepted by BLUMENFELD and PUTZIG (1914), as well as v. SAALFELD (1932), it is produced in breathing in consequence of periodical changes in the vagal tonus arising from the pulmonary branches of this nerve. The supporters of the other, so-called central theory (TRAUBE 1865 FRÉDERICQ 1913, HEYMANS 1929 and others) consider that the

vagal centre is under the direct influence of the respiratory centre, so that changes in the heart frequency and blood pressure arise parallel with the respiratory movements. Another theory is put forward by BAINBRIDGE (1920) according to which the pulse frequency is partly regulated by the heart itself. Thus with inspiration an increase of the pressure in the right half of the heart stimulates the nerve receivers in the right auricle, so that the pulse frequency increases. The consequence of expiration, when the pressure is decreased, is a slower pulse. According to NORDENFELT (1943) the Bainbridge reflex acts directly upon the activity of the sinus node and not on the tonus of the vegetative nerves.

To summarise this it may be said that at present it is impossible to give a general, comprehensive explanation of the rise of the respiratory arrhythmia. The different theories, however, do not seem to be mutually exclusive, and a co-operation of several factors, mechanical, nervous and humoral, is therefore not improbable.

In these days sinus arrhythmia is generally considered a physiological phenomenon. On the other hand, opinions are divided as to the extent to which pathological significance can be attributed to a high or low degree of it.

HEUBNER (1894) was one of the first to show that sinus arrhythmia, chiefly its respiratorily determined form, is specially distinct after different infectious diseases such as scarlatina, diphtheria and typhus. He interpreted the increased arrhythmia as an expression of a certain myocardial damage. However, as the increase was generally temporary he did not attribute to it any clinical importance. Later LOMMEL (1902) made similar observations after infectious diseases, and he considered that the increased arrhythmia was due to an increased lability in the vegetative nerve system. While the pulse was high in the fever stage, on the other hand, the arrhytmia was below normal. LUKOMSKI (1932) has observed an increased respiratory arrhythmia in children with acute rheumatic fever which he interpreted as due to an injury to the sinus node. The heart frequency in children with organic heart disease showed values which did not vary from the normal.

During recent years SECKEL (1934), MENGOLI (1937), NÁDRAI (1937—38), KLEMOLA (1942) and others have shown that the respira-

tory arrhythmia is specially distinct after diphtheria. Thus NÁDRAI, studying material composed of 50 children suffering from this disease, found a very definite respiratory arrhythmia in each case. This »convalescent arrhythmia» was so distinct that he distinguishes it as a special form of sinus arrhythmia. Yet the investigation showed that from the degree of the arrhythmia or the time of its appearance no direct conclusions could be drawn as to the condition of the heart muscle. On this point NÁDRAI agrees with MENGOLI's conception. According to NÁDRAI the occurrence of arrhythmia after infectious diseases is chiefly due to changes in the tonus of the vegetative nervous system.

MACKENZIE, who in 1902 arranged the first more comprehensive examination of pulse conditions in children, observed that sinus arrhythmia was least definite in the first years of life, while it increased towards puberty. The increase of the sinus arrhythmia with age was parallel to the decreasing pulse frequency towards puberty. On the basis of his investigations MACKENZIE concluded that the sinus arrhythmia is one criterion of a sound heart. His observations in this respect at different ages have lately been confirmed by SCHLOMKA (1937) who, by detailed electrocardiographic investigations, has demonstrated that sinus arrhythmia is most distinct during puberty and decreases successively with increasing age.

FRIBERGER (1912) registered the pulse curves of 312 children aged from 3—14 years, and in all cases observed a respiratory arrhythmia. As under normal conditions it was subjected to considerable variations he attributed no pathological importance to its different degrees.

WENCKEBACH and WINTERBERG (1927) reserve themselves as regards interpreting the sinus arrhythmia as one sign of a sound heart. According to these authors a definite sinus arrhythmia is not quite unusual in connection with organic heart disease but that a pathological importance can most often be attributed to an absence of sinus arrhythmia revealed by a completely regular pulse. Their opinion is accepted by SHERF and BOYD (1945).

According to NOUBÉCOURT (1936) and FEER (1941) a moderate degree of sinus arrhythmia is quite physiological, a higher degree of

it is mostly found in neurolabile children. The former emphasises that in interpreting arrhythmia in children one should in the first place consider its physiological form.

KÜHNE (1936) is of the opinion that respiratory arrhythmia chiefly depends on the pulse frequency. In examining children he could not demonstrate a respiratory arrhythmia if the pulse frequency exceeded 140 beats per minute. Children with heart diseases were no exception in that respect. As the occurrence of arrhythmia was conditioned solely by the pulse frequency, he considers that from the arrhythmia no conclusion can be drawn as to the condition of the heart muscle. His material, however, was rather small, comprising 47 children of whom 9 were normal.

SCHLOMKA and REINDELL (1936), on the basis of their observations on healthy sportsmen, consider that the respiratory arrhythmia is an expression of an appropriate adaptation of the heart's function. At the loading test the respiratory arrhythmia increased considerably above the normal. The test results were opposed to KÜHNE's conception mentioned above that the necessary condition for this arrhythmia's origin is a relatively low pulse, in spite of the fact that the heart frequency rose with the loading.

Most recently NORDENFELT (1943) has thrown light upon the details of respiratory arrhythmia under different conditions in children. By registering the electrocardiagram and the respiratory curves at the same time he observed in the age-group 3—15 years almost always a respiratory arrhythmia of which the intensity varied greatly. On the other hand in normal children under 1 year the sinus arrhythmia proved not to be dependent on the breathing. Comparative observations of the respiratory arrhythmia in children suffering from organic heart disease or from different kinds of infectious diseases showed no palpable differences from normal conditions. Like SCHLOMKA and REINDELL, NORDENFELT observed a considerable increase of sinus arrhythmia after exertion. As it seems to vary normally within wide limits in children — even in the same individual — NORDENFELT considers that one is seldom entitled to draw any direct conclusions from this arrhythmia regarding the condition of the heart.

Sinus arrhythmia has been studied in a total of 300 children, of whom 200 were normal. Of these latter the children under 1 year were placed at my disposal throught the kindness of Dr LIND. The pathological material was composed of 50 children with congenital heart disease and 50 with rheumatic heart affection.

In calculating the sinus arrhythmia the so-called frequency index, RI, has been used, which is a mathematical expression of the degree of arrhythmia. The formula, introduced by SCHLOMKA and REINDELL (1936), is as follows:

$$RI = \frac{100 \cdot \left[\sum_{o}^{n} p\,max. - \sum_{o}^{n} p\,min. \right]}{n \cdot Pm}$$

According to this formula RI is obtained by first calculating the total of a number (n) of maximum pulse intervals (p max.). From this total is deducted the total of an equal number of the shortest pulse intervals (p min.). The difference thus obtained is then expressed in a percentage of the corresponding mean pulse time (Pm). SCHLOMKA and REINDELL propose that n should be 3 or 4. As the length of the curves in some of my investigations only allowed the observation of 3 maximum and minimum pulse intervals this number for n has been used in all the cases.

Normal cases

The normal material is divided into the age-groups 0—1, 1—5, 6—10 and 11—15 years, each containing 50 children. Both as regards age and sex the material was divided as equally as possible within the different groups. All the children showed normal heart by physical examination. In no case have such infectious diseases as rheumatic infection, scarlatina and diphtheria occurred in the case history, such as could be thought to have influenced the organ of circulation. As regards haemoglobin, red blood corpuscles and sedimentation rate, the values were within the normal limits. This was also true of the blood pressure, which was measured in the children

TABLE 1.

Sinus arrhythmia in normal children.

RI = frequency index (SCHLOMKA), Pm = pulse frequency (beats per minute) M = mean, σ = standard deviation, ε (M) = standard error of the mean.

Age in years	Number of cases	Boys	Girls	RI		Pm	
				M $\pm$ ε (M)	σ	M $\pm$ ε (M)	σ
0—1	50	26	24	10.9$\pm$0.71	5.07	140$\pm$4.09	28.89
1—5	50	26	24	11.2$\pm$3.91	3.91	105$\pm$2.88	20.42
6—10	50	23	27	13.5$\pm$0.68	4.82	89$\pm$3.69	26.13
11—15	50	27	23	15.1$\pm$0.68	4.78	79$\pm$2.24	15.84

over 2 years old. In the phonocardiographic examination of children over 1 year old, in no case was a murmur registered with a frequency exceeding 250 cycles per second. According to MANNHEIMER's investigations such murmurs are within physiological limits.

From Table 1 it appears that sinus arrhythmia was observed in all the children. Its degree rose with the age in such a way that the frequency index for those under 1 year was 10.9 $\pm$ 0.71, while the corresponding figure in the age-group 11—15 years, was 15.1 $\pm$ 0.68. This increase ran parallel with a decreasing pulse frequency as the age increased. Thus the figures for this frequency were 140 $\pm$ 4.09 and 79 $\pm$ 2.24 beats per minute in the age-groups 0—1 and 11—15 years respectively. The oldest group showed the least deviation (δ = 15.84) as regards the values for pulse frequency. No difference connected with the sex in the sinus arrhythmia and the pulse frequency could be observed in the different groups.

Pathological cases

Patients with congenital heart disease were 50 in number aged 6—10 years. In most of the cases they had had the disease from their earliest years and there was no information in the case history to show that they had acquired it. In the material there were 9 cases of morbus coeruleus. Further, 8 cases of patent ductus Botalli in which the diagnosis was later verified at an operation. Of these

TABLE 2.

Sinus arrhythmia in children with congenital heart disease and with rheumatic heart disease.

RI = frequency index (SCHLOMKA), Pm = pulse frequency (beats per minute)
M = mean, σ = standard deviation, ε (M) = standard error of the mean

Cases	Age in years	Number of cases	Boys	Girls	RI		Pm	
					M $\pm$ ε (M)	σ	M $\pm$ ε (M)	σ
Congenital heart disease	6—10	50	31	19	7.2$\pm$0.41	2.90	95$\pm$2.99	21.19
Rheumatic heart disease	6—10	50	22	26	6.1$\pm$0.39	2.81	97$\pm$3.08	21.74

8 patients 7 had a normal electrocardiogram. All the other children showed pathological changes in the electrocardiogram, as only definite cases of congenital heart disease had been chosen. A phonocardiographic examination was made in 43 cases, and of them 40 had murmurs with a frequency up to 400—1000 cycles per second. MANNHEIMER's investigations indicate that these high frequency murmurs are in the majority of cases pathologically and organically conditioned, and support a suspected heart disease. Only in 3 cases was a physiological murmur registered up to 250 cycles per second.

The other group of pathological cases was composed of 50 children aged 6—10 years with heart disease in association with rheumatic fever. All these cases showed an increased sedimentation rate and typical alterations in the electrocardiogram (mostly prolonged conduction time, changes in the S—T segments or isoelectric or negative T waves in at least two leads).

In the phonocardiographic examination of 45 of the patients a physiological murmur was registered in 38 of them, in most cases up to 250 cycles per second. In 7 of the patients higher frequency ranges were observed up to 400—500 cycles per second. In these latter cases the rheumatic infection was in the nature of a relapse.

The pathological material was also selected as far as possible so that there was an equal number of the different ages in each group.

TABLE 3.

Sinus arrhythmia.

The difference between the means of SCHLOMKA's frequency index under normal and pathological conditions.

D = difference between two means
ε (D) = standard error of the difference

Cases	Age in years	D ± ε(D)
Normal children	0—1	0.3±0.89
» »	1—5	2.3±0.87
» »	6—10	1.6±0.96
» »	11—15	
» »	0—1	2.6±0.98
» »	6—10	
» »	0—1	4.2±0.98
» »	11—15	
» »	1—5	3.9±0.88
» »	11—15	
Children with congenital heart disease	6—10	6.3±0.79
Normal children	6—10	7.4±0.78
Children with rheumatic heart disease	6—10	
Children with congenital heart disease	6—10	3.7±0.82
Normal children	0—1	6.3±0.79
Children with rheumatic heart disease	6—10	

The results collected in Table 2 indicate a frequency index of 7.2 ± 0.41 for children with congenital heart disease, while the corresponding figure for children with rheumatic heart disease was 6.1 ± 0.39. Cases of morbus coeruleus in the former group showed a tendency to relatively low index values (the average was 6.2).

The mean pulse time in both groups was 95 ± 2.99 and 97 ± 3.08 beats per minute respectively.

For more detailed gauging of the sinus arrhythmia in these cases the results of the tests are compared in Table 3.

First, as regards sinus arrhythmia under normal conditions, the results indicate no certain difference in the figures for the frequency index in children of neighbouring age-groups. There, is however, a probable difference between the age-groups 1—5 and 6—10 years. On the other hand, going further, there is probably a difference between the frequency indices of the children in the age-groups 0—1 and 6—10; yet the difference is definite between the age-groups 0—1 and 11—15, as well as between the age-groups 1—5 and 11—15 years. This signifies, in other words, that sinus arrhythmia is more distinct in later childhood than in the first years of life.

A comparison of the results shows that sinus arrhythmia is less pronounced in the pathological cases. Table 3 shows that this difference is confirmed not only in relation to the figures in the corresponding age-group of the normal material but also in relation to the lowest normal figures shown by children in the age-group 0—1 year.

Of all the cases those with rheumatic heart disease showed the lowest frequency index. Yet the difference is not statistically confirmed in comparison with children suffering from congenital heart disease.

Discussion. The examinations have shown that sinus arrhythmia is most distinct in the later years of childhood. The results confirm MACKENZIE's observations in this respect. SCHLOMKA, who preferably studied this subject in adults, obtained a frequency index varying between ca 4 and 12, thus a lower figure than in the children examined as above. In one group of children aged 6—15 SCHLOMKA obtained, as NORDENFELT did later, a figure of ca 17, which is somewhat in excess of my corresponding figures.

As regards the explanation of the relatively strongly marked sinus arrhythmia in later childhood it is considered to be partly conditioned by the decreasing pulse frequency towards puberty. The results of the measuring of frequency in the tests tend to support this assumption, as the increase of sinus arrhythmia with increasing age was parallel with a decrease of the pulse frequency. However, it is probable that this increase is due to several factors. Knowing that the rise of sinus arrhythmia is connected with varia-

tions in the tonus of the extracardiac nerves, there is reason to assume that the relatively high sinus arrhythmia during puberty is partly connected with the lability in the vegetative nerve system which is considered to characterise these years.

The tests have shown a definite decrease of the sinus arrhythmia in children with congenital heart disease and with rheumatic heart affection. SCHLOMKA made simlar observations in adults with decompensated heart disease. As far as I know, no examination of a larger number of children has yet been made.

Knowing the complicated mechanism of the rise of the sinus arrhythmia it seems difficult to judge the reason for its decrease in the pathological children. If the question is judged against the background of KÜHNE's observations, according to which a low pulse frequency involves a higher degree of sinus arrhythmia, it seems that in this case the conditions are not so simple. Thus, the pulse frequencies both in children with congenital heart disease and those with rheumatic heart affections (95 ± 2.99 and 97 ± 3.08 beats per minute respectively) agree in the main with the figures in the corresponding age-groups of normal children (89 ± 3.69 beats per minute). Further it should be noted that in the pathological children the sinus arrhythmis was less also in relation to the normal children in the age-group 0—1 year, who showed the highest pulse frequency (140 ± 4.09 beats per miute).

As the stimulus normally proceeds from the sinus node it may be considered theoretically that the decrease of the sinus arrhytmia is due to some damage to the node which decreases its sensitiveness to external impulses. In this connection the theory may be mentioned, supported by WEBER (1937), according to which the sinus node contains a »Kontraktionssubstanz», necessary for its normal function. This »substance» is said to be easily affected by different injurious factors. As regards the decreased sinus arrhythmia in children with rheumatic heart affection the possibility of an injury to the sinus node seems not impossible, knowing that this disease is often accompanied by limited infectious foci in the heart. In respect also of the origin of the low sinus arrhythmia in congenital heart disease a congenital lowering of the sinus node's sensitiveness can be theoretically assumed.

For different explanations of a lowered sinus arrhythmia the importance of various humoral factors can also considered. Thus for example, the carbonic acid in the blood is supposed to have a direct effect on the tonus of the vegetative nerve system and possibly also on the function of the sinus node. An observation made by Koch (1936/37) that the sinus arrhythmia decreases in suffocation may be mentioned here. As there is justification for assuming that a decrease of the heart's functional ability, not least in cases of congenital diseases, leads to an increased carbonic acid tension in the blood, it may be thought that this increase may lower, directly or indirectly, the sensitiveness of the sinus node and thus result in a decreased sinus arrhythmia.

Summarising the above it may thus be said that on the basis of statistical calculations it appears clear that the sinus arrhythmia in children increases with age. Besides this it seems to be influenced also by the condition of the heart. Thus a decreased sinus arrhythmia was observed in children with congenital heart disease and rheumatic heart affection.

Extrasystoles

Extrasystoles are premature heart contractions which disturb the normal rhythm. The basis of such premature beats is the fact that every specific fibre of the heart has the ability to form stimuli autonomously. Under normal conditions the stimulus formation in these ectopic centres is subdued by the impulses from the sinus node which works with the highest frequency activity within the specific muscular system. If, on the other hand, the intensity of the stimulus formation in the sinus node is decreased, or if the activity in an ectopic centre is increased the latter will dominate. SHERF and BOYD throw light upon the question quite simply in the following way: »The centre, which forms stimuli at the highest rate, controls the heart.«

As regards the origin of the extrasystoles one differentiates a supraventricular and a ventricular form. A middle position is occupied by atrio-ventricular extrasystoles proceeding from Tawara's node. These however, seem to be generally referred to the supraventricular.

The extrasystoles can appear irregularly or can be in a definite relation to the normal beats (e.g. bigeminy).

The extrasystoles occur less often in children than in adults. Thus COWAN and RITCHIE (1922) found in 226 patients with extrasystoles only 7 who were under 19 years. All age-groups were included in his material. SMITH (1924) relates that in similar examinations he found only 2 children, 11 and 12 years old, in 100 patients with this arrhythmia.

Information regarding the frequency of extrasystoles in children varies greatly. VISCO who, in 1912 examined 1000 children at a

hospital, found extrasystoles in 48 cases. Two of the children were quite free of other symptoms while the rest suffered from undernourishment of infectious diseases of different kinds. Yet he did not attribute any noteworthy clinical importance to this form of arrhythmia.

In FRIBERGER's material, comprising 321 healthy children, there were 2 cases of extrasystoles.

HECHT (1913) states that extrasystoles are extremely rare, especially during early childhood. In 7 children, 10 weeks to 11 years old, they appeared in connection with infectious diseases, such as pneumonia, tuberculosis and diphtheria. He considers that extrasystoles of supraventricular origin indicate a more serious disturbance than those of ventricular.

In examining 42 children aged 1 day to 14 years KRUMBHAAR and JENKS (1917) found none with extrasystoles. ANTELL (1931), whose material consisted of 400 children under treatment in hospital, came to the same conclusion.

HALSEY (1917) discovered only 3 patients with extrasystoles among 92 children suffering from different kinds of heart disease.

PERRY (1931) who examined 100 healthy school children, found this form of arrhythmia in only 1 case.

SHOOKHOFF and TARAN (1931), in an electrocardiographic examination of 259 healthy children aged 6—14 years, found 2 cases of extrasystoles. In a similar examination of 100 healthy children, HAFKESBRING, DRAWE and ASHMAN (1931) state that they discovered this form of arrhythmia in 2 cases, and in a group of 100 children with rheumatic heart disease they found only 1 with extrasystoles. In a similar number of children with congenital heart disease there were none with this form of arrhythmia.

Extrasystoles after scarlatina are fairly rare in children. Such cases have been described by WICKSTRÖM (1933), BLUMBERGER (1936), also by v. KISS and ROMHÁNYI (1938).

After diphtheria on the other hand extrasystoles seem to be more usual. Thus KLEMOLA (1942), in material composed of 41 convalescents from diphtheria — most of them under 20 years — found extrasystoles in 11 cases. At a loading test extrasystoles were provoked in 18 of the patients.

The most comprehensive examinations of extrasystoles in children have been carried out by LYON and RAUH (1939). In a simple physical examination of 5114 new-born children they found extrasystoles in only 3 cases. This figure is probably somewhat too low, as the arrhythmia here in question is often difficult to demonstrate by simple physical examination alone (BURGHARD and WUNNERLICH 1930). LYON and RAUH found extrasystoles in 59 cases among 2672 children with sound hearts (2.2 %), 20 in 468 children with heart disease (4.3 %), a somewhat higher frequency. Among 1782 school-children sent to hospital because of suspected heart disease 20 cases of extrasystoles were found (1.1 %).

BURGHARD and WUNNERLICH maintain that extrasystoles appear relatively often in new-born children. In 32 such children they discovered 10 cases. SEHAM (1921) and NÁDRAI (1941) have, on the other hand, like LYON and RAUH, not found this form of arrhythmia more often during the period immediately after birth than in older children.

The cases reported above show that it is often difficult to ascertain what etiological factors co-operate in causing the extrasystoles. According to a conception held by LECONTE (1911), HESS (1913) and NOUBÉCOURT (1939), of predisposing causes in children the most important are organic heart disease, hypertension, toxic effects of certain drugs, e.g. digitalis, coffee and tobacco, gastro-intestinal affections and neurolability. It seems however, that many of these factors have seldom any direct practical application in childhood. The question is illuminated by LYON and RAUH who in their work have examined 79 children with extrasystoles just to discover possible predisposing factors. Here they found congenital heart disease and rheumatic heart disease in 7 and 11 patients respectively. Two of the children had had diphtheria and 1 scarlatina. In the other 58 children no symptoms of disease could be demonstrated, nor was there any information regarding earlier illnesses which could be thought to have given rise to the extrasystoles. In 2 of the children neurolability might be considered as a predisposing factor. Disturbance of the digestive apparatus, which is given as a predisposing factor by some authors (PAL 1936), was very seldom found in the children of this material.

According to SIEGEL (1945), extrasystoles in children are more frequently due to an infection of the heart musculature or to a general infection of the organism.

Finally it can be mentioned that ANTELL (1931) includes a hereditary component in the occurrence of extrasystoles.

Extrasystoles can exist for a long time without causing the patient any direct trouble. SMITH (1927) mentions 2 boys, 11 and 12 years old, in whom this form of arrhythmia lasted ½ and 2 ½ years respectively. BASS (1926) describes 7 children aged 3—8 years who showed extrasystoles for a period varying between 1 and 4 ½ years. In 5 of the children the heart function was normal in all other respects. PEIPER (1926) has followed the development of 12 children 2—18 years old in whom the extrasystoles remained from 1 week to 5 years. Eleven of them remained free from heart disease during the period of observation. None of these children showed any kind of insuffiency symptom. The same observations were later made by LEFFKOWITZ (1931) in a group of 26 children in whom the extrasystole was apparent for a period of 1 ½—2 ½ years.

In the material used by LYON and RAUH 42 children aged 1 month to 10 years were examined regularly for a period of up to 3 ½ years. Thirteen of them showed extrasystoles on only one occasion, in 14 cases the arrhythmia remained 1 to 6 months, in 10 cases 6 months to 2 years, and in 5 cases more than 2 years. Of all the children 24 had rheumatic or congenital heart disease while the rest were apparently healthy. As regards the duration of the extrasystole there was no direct difference between the children in the different groups. The prognosis was in all cases good and the general impression was that the extrasystoles did not affect either the heart function or the growth and development of the children.

In my material extrasystoles was found in 86 cases (1.53 %). The material was divided as regards the origin of the extrasystoles. Division was sometimes a question of interpretation because the transition between the types in the different groups is often indistinct.

TABLE 4.

Extrasystoles.

	Boys	Girls	Totals	Origin of the extrasystoles	Number of cases
Supraventricular extrasystoles	21	13	34	Auricular	16
				Nodal	15
				Various	3
Ventricular extrasystoles	28	24	52	Right type	31
				Left type	11
				No axis deviation	8
				Not localised	2
Totals	49	37	86		86

The division of the material (Table 4) shows that in 34 children (21 boys) the extrasystoles were of supraventricular and in 52 children (28 boys) of ventricular origin. In the former group the auricular extrasystoles have been distinguished from the nodal extrasystoles proceeding from Tawara's node. The auricular form consisted partly of sinus extrasystoles, partly of those in which the stimulus formation proceeded from other parts of the auricle. Sinus extrasystoles was found in 4 children and were characterised by an extra beat with a normal ventricular complex preceded by a normal P-wave and with the same conduction time as in the normal beat (Fig. 1.).

When a normal ventricular complex is preceded by a P-wave, negative at times, and the P—Q interval is longer or somewhat shorter than in the normal beat, it has been regarded as an auricular extrasystole proceeding from outside the sinus node (Fig. 2.).

The nodal extrasystoles have been divided into 3 groups according to the part of Tawara's node in which the ectopic stimulus formation can be thought to take place. Those in which a ventricular complex is preceded by a P-wave, sometimes negative, and in which the conduction time is considerably shortened in relation to the normal beat, have been regarded as extrasystoles arising from the upper part of Tawara's node (Fig. 3.).

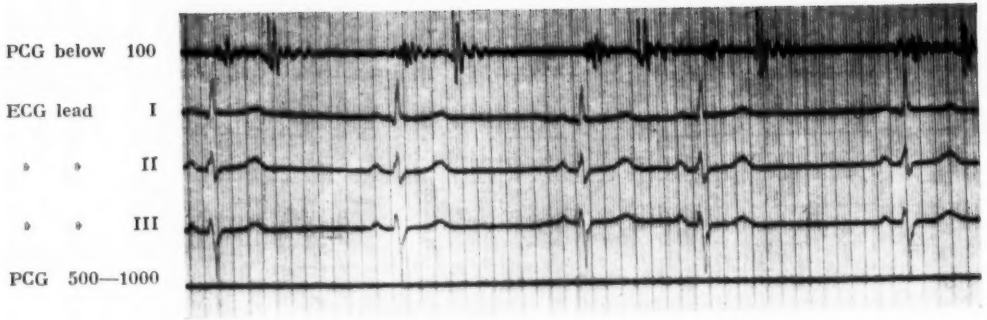


Fig. 1. Sinus extrasystole in a boy of 10 years.

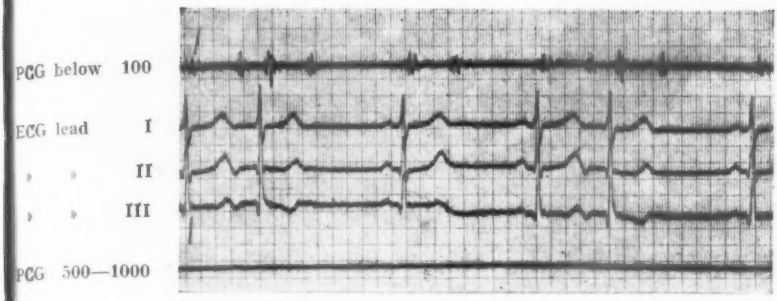


Fig. 2. Auricular extrasystoles in a boy of 10 years and 8 months.

The extrasystoles arising from the middle part of Tawara's node — nodal beats in a more limited sense — have been distinguished by the fact that the auricular wave is included in a normal ventricular complex (Fig. 4.).

Finally, if an extra beat with a normal QRS complex in close connection is followed by a P-wave, this has been interpreted as an extrasystole arising in the lower part of Tawara's node (Fig. 5).

The ventricular extrasystoles are characterised both by an absence of the auricular wave and by a pathological ventricular complex. As there are still different opinions as to the interpreta-

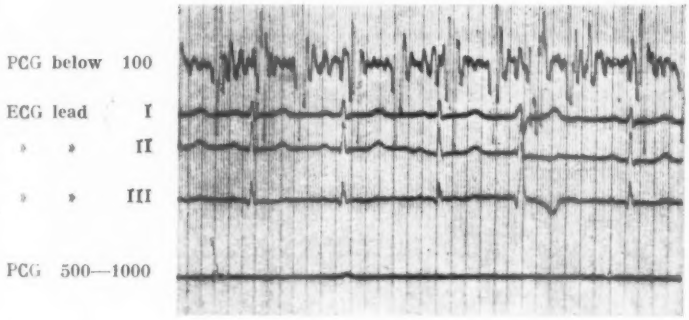


Fig. 3. Extrasystole originating in the upper part of Tawara's node in a boy of 12 years.

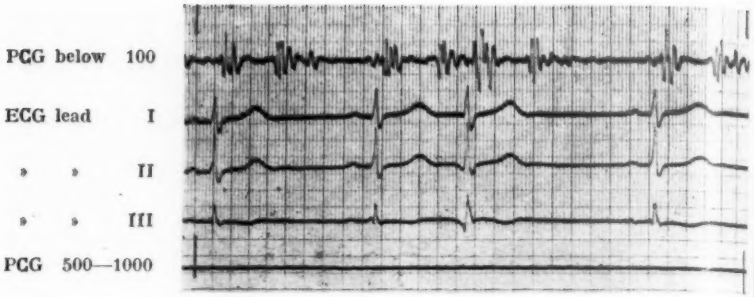


Fig. 4. Extrasystole originating in the middle part of Tawara's node in a boy of 7 ½ years.

tion of the localisation of stimulus formation on the basis of the appearance of the ventricular complex, it has seemed advisable to distinguish only 2 main groups of ventricular extrasystoles according to the direction of the electrical axis. In so doing those in which the electrical axis — calculated from EINTHOVEN's triangle — was above 90° have been termed ventricular extrasystoles of the right type. (Fig. 6).

The corresponding figure for ventricular extrasystoles of the left type was below 0° (Fig. 7.).

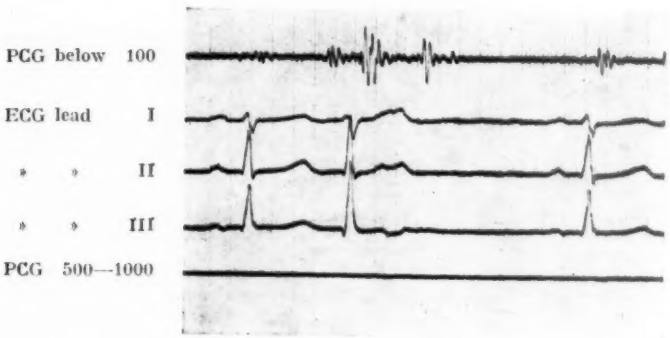


Fig. 5. Extrasystole originating in the lower part of Tawara's node in a boy of 9 years and 8 months.

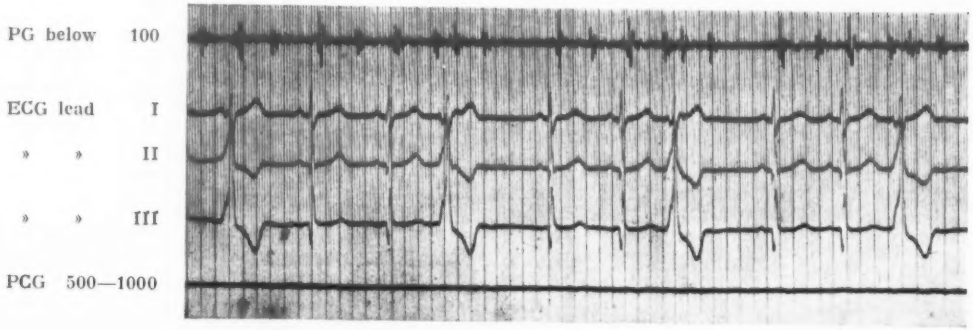


Fig. 6. Trigeminy. Ventricular extrasystoles of right type in a girl of 2 ½ years

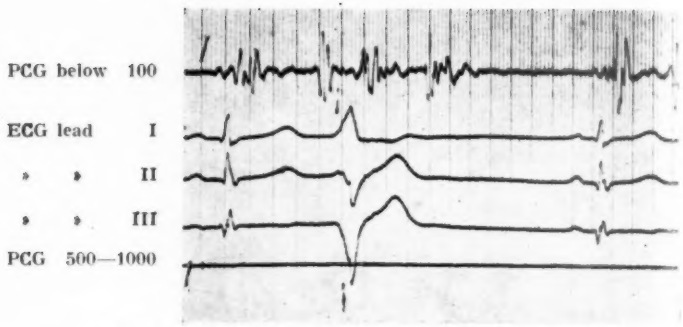


Fig. 7. Ventricular extrasystole of left type in a boy of 12 years.

Among the ventricular extrasystoles 2 cases were classed as »not localised», because they were registered only in lead II. In these cases the examinations were made with a one-lead-electrocardiograph.

Supraventricular extrasystoles

The 34 cases of supraventricular extrasystoles are collected in Table 5. From this it appears that only 8 of the children were under 5, while the rest are distributed in the age-groups 5—15 years. In the group 0—1 year there is a premature child, 7 weeks old. The material contained 21 boys and 13 girls.

Of the information in the case histories special attention was paid to previous —i.e. in the preceding 2 years — infectious diseases such as scarlatina, diphtheria, rheumatic infection and repeated tonsilitis. Seven of the children had had some of these illnesses while the rest had been healthy. When examined only 2 of the children were suffering from active infectious diseases (rheumatic fever and tonsilitis); 12 of them were stated to have had subjective troubles, chiefly dyspnoea and stitch in the region of the heart. Neurolability was found in 8 cases. On the occasion of the examination all the children were in good general condition.

TABLE 5.
Supraventricular extrasystoles.

TABLE 5.

Supraventricular extrasystoles.

Age group Yrs	Cases			Previous infectious diseases				Active infectious disease			Symptoms								Blood pressure (mm Hg)			Phonocardiogram Systolic murmur (cycles per second)			
	Boys	Girls	Total	Scarlet fever	Rheumatic fever	Tonsillitis	none	Rheumatic fever	Tonsillitis	none	General condition good	Dyspnoea	Stitch	Neurolability	Various	No symptoms	95—105	105—115	115—125	Up to 175	Up to 250	Up to 400	Up to 500
0—1	3	2	5	—	—	—	5	—	—	5	5	1	—	—	1	3	—	—	—	—	—	1	—
1—5	2	1	3	—	—	—	3	—	—	3	3	—	—	1	—	3	—	1	—	—	1	1	—
6—10	7	6	13	2	1	—	13	—	—	13	13	3	1	3	1	5	4	4	3	—	12	—	1
11—15	9	4	13	1	2	1	11	1	1	11	13	4	1	4	—	6	3	3	3	1	7	2	1
Totals:	21	13	34	3	3	1	32	1	1	32	34	8	2	8	2	17	7	8	6	1	20	4	2

Age group Yrs	Pulse frequency (beats per minute)				P-Q (seconds) normal beats			Angle of electrical axis (degrees)					»Myocardial damage«	Origin of the extra systoles				Allorhythmias	
	60—80	80—100	100—120	120—140	140—160	< 0.10	0.10—0.15	0.15—0.17	+30—+50	+50—+70	+70—+90	+90—+100	+100—+130		Auricular	Tawara's node upper part	Tawara's node middle part	Tawara's node lower part	Various	Bigeminy	Trigeminy
0—1	—	1	—	—	4	5	—	—	—	1	1	—	3	1	2	1	1	—	1	1	—
1—5	—	—	3	—	—	—	3	—	1	2	—	—	—	—	1	1	—	—	1	—	—
6—10	2	11	—	—	—	—	9	4	1	4	4	—	—	3	7	1	2	2	1	—	1
11—15	2	10	1	—	—	—	10	3	1	9	2	1	—	1	6	4	2	1	—	—	—
Totals:	4	22	4	—	4	5	22	7	3	16	7	5	3	5	16	7	5	3	3	1	1

The blood pressure was measured in 21 cases but no deviation from the normal was observed.

When 27 of the children underwent a phonocardiographic examination, 6 of them showed a murmur, probably organically conditioned, with a frequency up to 400—500 cycles per second. In the others the registered murmur was within physiological limits.

The pulse frequency was normal in all cases. This was also true of the conduction time, the limits of which were 0.08 and 0.17 second.

As regards the direction of the electrical axis 5 children in the age-group 6—15 years showed a relative right axis deviation 90° to + 100°. Three children under 6 months showed figures between 100° and 130°, which, however, lie within the physiological limits for those years (MANNHEIMER 1940). In all the other cases no axis deviation could be observed.

Signs of »myocardial damage» — such as pathological initial deflexions, changes in the S—T segments, negative or iso-electric T-waves in at least two leads — were found in 5 cases.

The more detailed division of the material as regards the origin of the extrasystoles appears from the tables.

Allorhythmias were found in 2 cases (bigeminy and trigeminy).

Ventricular extrasystoles of right type

Ventricular extrasystoles of right type were found in 31 children, all of them more than 1 year old (Table 6.).

Of all the children 15 were boys and 16 girls. Previous infectious diseases were in 2 of them rheumatic infection and in 1 scarlatina, while the rest had been healthy. When examined 2 of the children were suffering from active infectious disease (rheumatic infection). The general condition was good in all cases. Only 2 of the children had subjective trouble such as dyspnoea and stitch in the region of the heart. Neurolability was found in 5 cases.

The blood pressure was measured in 24 cases, and 4 of them in the age-group 6—15 years showed somewhat increased values

TABLE 6.
Ventricular extrasystoles of right type

TABLE 6.

Ventricular extrasystoles of right type.

Age group Yrs	Cases			Previous infectious diseases			Active infectious disease		Symptoms					Blood pressure (mm Hg)					Phonocardiogram Systolic murmur (cycles per second)		
	Boys	Girls	Total	Scarlet fever	Rheumatic fever	None	Rheumatic fever	None	General condition good	Dyspnoea	Stitch	Neurolability	No symptoms	90—100	100—110	110—120	120—130	130—140	Up to 175	Up to 250	Up to 400
1—5	1	5	6	—	1	5	1	5	6	—	—	—	6	2	—	—	—	—	—	6	—
6—10	9	4	13	1	—	12	—	13	13	1	—	3	9	—	1	7	3	1	2	9	2
11—15	5	7	12	—	1	11	1	11	12	—	1	2	11	—	2	1	4	3	—	11	1
Totals:	15	16	31	1	2	28	2	29	31	1	1	5	26	2	3	8	7	4	2	26	3

Age group Yrs	Pulse frequency (beats per minute)				P-Q (seconds) Normal beats			Angle of electrical axis (degrees)											»Myocardial damage«	Allorhythmias		
								Normal beats						Extrasystoles								
	60—80	80—100	100—120	120—140	< 0.10	0.10—0.15	0.15—0.19	—30	+30—+50	+50—+70	+70—+90	+90—+100	+100—+110	+95—+100	+100—+110	+110—+120	+120—+140	+140—+160		Bigeminy	Trigeminy	Quadrigeminy
1—5	—	1	4	1	1	4	1	—	2	2	1	—	1	—	5	—	—	1	1	1	1	—
6—10	4	6	3	—	1	11	1	1	1	6	5	—	—	—	8	3	2	—	1	1	—	—
11—15	2	8	2	—	1	7	4	—	2	3	5	1	1	3	7	1	1	—	—	2	—	1
Totals:	6	15	9	1	3	22	6	1	5	11	11	1	2	3	20	4	3	1	2	4	1	1

(130—140 mm Hg.). In the other cases the figures were within normal limits.

All the children were examined phonocardiographically. In 3 of the children a murmur was registered with a frequency up to 400 cycles per second, probably organically conditioned.

The figures for pulse frequency and conduction time were in all cases within normal limits.

As regards the direction of the electrical axis of the normal beats, 27 of the children showed no relative axis deviation. In 1 case the electric axis had a left position (—30°), and in 3 cases a right position (+ 90° to + 110°). As regards the extrasystoles the majority (24) showed values between + 100° and + 120°.

Signs of »myocardial damage» — prolonged conduction time, changes in the QRS complexes, S—T intervals or in T-waves — were found in only 2 children.

Of allorhythmias there were 4 cases of bigeminy, 1 case of trigeminy and 1 of quadrigeminy.

Ventricular extrasystoles of left type and those without relative axis deviation

In Table 7 the 21 children are collected with ventricular extrasystoles differing from the right type. The sex distribution shows a slight predominance of boys (13). None of the children had previously had any infectious disease, and none showed any signs of such when examined.

In all the children the general condition was satisfactory. Only 3 of them had subjective trouble such as dyspnoea and fatigue. Neurolability was found in 4 cases.

No pathological values were measured in the blood pressure.

When phonocardiographically examined 3 out of 18 children showed a systolic murmur with a frequency up to 400—500 cycles per second, probably organically conditioned.

The pulse frequency showed in general normal figures. In 1 boy of 13 years with neurolability a tachycardia of 160 beats per minute was observed.

TABLE 7.
Ventricular extrasystoles of left type and with no axis deviation

TABLE 7.

Ventricular extrasystoles of left type and with no axis deviation.

Age group Yrs	Cases			Previous infectious diseases	Active infectious disease	Symptoms					Blood pressure (mm Hg)				Phonocardiogram Systolic murmur (cycles per second)			
	Boys	Girls	Total			General condition good	Dyspnoea	Various	Neuro-lability	No symptoms	90—100	110—120	120—130	130—140	Up to 175	Up to 250	Up to 400	Up to 500
1—5	4	2	6	—	—	6	—	1	2	4	1	3	—	—	2	2	1	—
6—10	7	3	10	—	—	10	1	1	—	8	—	5	2	—	—	6	1	1
11—15	2	3	5	—	—	5	—	2	—	3	—	2	1	2	—	5	—	—
Totals:	13	8	21	—	—	21	1	4	2	15	1	10	3	2	2	13	2	1

Age group Yrs	Pulse frequency (beats per minute)				P-Q (seconds) Normal beats				Angle of electrical axis (degrees)																«Myocardial damage»	Allo-rhythmias	
									Normal beats						Extrasystoles												
	60—80	80—100	100—120	140—160	< 0.10	0.10—0.15	0.15—0.18	0.22	—30	+30—+20	+50—+40	+60—+50	+80—+60	+90—+80	—60	—40—60	—20—40	±0—20	+40—20	+60—+40	+80—+60	+90—+80			Bigeminy	Trigeminy
1—5	—	1	4	1	1	5	—	—	—	—	1	—	4	1	—	1	1	2	—	—	1	1	—	—	—	—
6—10	3	6	—	—	1	4	4	1	1	3	1	3	—	1	1	2	1	1	1	1	1	—	2	—	—	—
11—15	—	3	2	1	—	4	1	—	—	—	—	3	3	—	—	2	—	—	—	—	3	—	—	2	2	
Totals:	3	10	6	2	2	13	5	1	1	3	2	6	7	2	1	5	2	3	1	1	5	1	2	2	2	

In 1 case an increased conduction time (0.22 second) was noted, otherwise low values within normal limits (0.09—0.18 second.).

The electrical axis of the heart, measured from the normal beats, showed, except in one child, no relative axis deviation (+20° to + 90°). In this case the axis deviation was left (—30°). As regards extrasystoles there were 11 cases with relative left axis deviation (—15 to —60°), and 8 cases without deviation (+20 to 90°). In 2 cases the electrical axis could not be determined as the extrasystoles were only registered in lead II.

Signs of »myocardial damage» — prolonged conduction time, changes in the QRS complexes, S—T intervals or the T-waves — were observed in 2 children.

Allorhythmias were found in 4 cases, bigeminy and trigeminy.

Repeated examinations of children with extrasystoles

Control examinations have been made on 32 of the 86 children with extrasystoles mentioned above (Table 8 A—B). In 9 cases they were of supraventricular type and in 23 cases of ventricular origin. The period of observation varied between 2 weeks and 4 ½ years.

Table 8 A shows that of the 9 children with supraventricular extrasystoles 4 showed this form of arrhythmia at one examination only. In the other 5 cases the extrasystoles were still there 3—7 months after their first registration. Two of the 9 children were newborn infants (the one premature). In both cases the arrhythmia was diagnosed prenatally. These observations agree quite well with the examinations of BURGHARD and WUNNERLICH, from which they pointed out that the extrasystoles which appear in the first days of life usually disappear within the next 2 weeks. Of possible predisposing factors in the origin of the supraventricular extrasystoles there was in 1 case an organic heart disease and in 2 cases a previous or active rheumatic affection. No direct difference in the duration of the extrasystoles could be observed between the cases.

TABLE 8. *Repeated examination of children with extrasystoles.*

A. *Supraventricular extrasystoles.*

Age group Yrs	Number of cases	Months							Years	Possible predisposing causes of ES
		1	2	3	4	6	7	8	2 ½	
< 1	1	N								Premature
	2	N								New-born
1—5	3					ES				Organic heart disease
	4			ES						
6—10	5				ES					
11—15	6							N		Rheumatic heart disease 1 ½ years previously
	7	ES			ES		ES			
	8			ES			ES			
	9	N	N	N	N				N	Active rheumatic fever

B. *Ventricular extrasystoles*

Age group Yrs	Number of cases	Weeks	Months					Years						Possible predisposing causes of ES
		2	1 ½	2	2 ½	4	8	1	2	2 ½	3	3 ½	4 ½	
1—5	1			ES										
	2						ES							
	3	ES												Acute rheumatic fever
	4											ES		
	5							ES						
6—10	6										ES		ES	
	7							ES						
	8		N											Organic heart disease
	9			ES						ES				
	10							N						
	11							N						
	12				ES	N						N		Repeated tonsilitis
	13		ES											
	14						N							Scarlet fever 1 year before
	15			N										
	16							ES	ES					
11—15	17								N	N				Acute rheumatic fever
	18	ES												
	19					N								
	20				ES									
	21								ES					
	22										ES			
	23							N						

ES = extrasystoles N = normal rhythm

Of the 23 children with ventricular extrasystoles 8 showed this form of arrhythmia at one examination only (Table 8 B). In 1 case (no. 12) it disappeared within 4 months. In the other 14 children the extrasystoles remained during the observation period varying between 2 weeks and 4 ½ years. In 18 cases predisposing factors which might have caused the ventricular extrasystoles were lacking. One child had organic heart disease and the other 4 had infectious diseases of different kinds. As regards the duration no direct difference could be observed between the cases. In cases nos. 16 and 21, there was bigeminy and trigeminy respectively which remained unchanged during the period of observation.

All the children showed a satisfactory general condition at the examinations. In no case was there any sign of circulation disturbances. During the period of observation the children seemed not to be more sensitive than usual to different illnesses. Thus only 1 child (no. 11) had had an infectious disease (scarlatina), 4 months before the control examination.

It is worth noticing that in those cases where the extrasystoles remained, the position of the electrical axis did not undergo any change on the whole.

About half of the children had been given chinidin or strychnine. No difference in regard to the duration of the extrasystoles could be observed between the treated and untreated cases.

Discussion. Extrasystoles were found in a total of 86 children. Of these 5 were under 1 year, 15 between 1 and 5 years, 36 between 6 and 10, and 30 between 11 and 15. The sex division showed a slight predominance of boys (49). In the material of Lyon and Rauh, comprising 42 children with an extrasystole, there were 18 boys and 24 girls.

The division of the material in respect of the origin of the extrasystoles showed in 34 cases a supraventricular (15 nodal) and in 52 cases a ventricular type. The observation that the latter is the most usual in children confirms Lyon and Rauh's investigations. Their material included a series of 22 children with registered extrasystoles and of them 16 showed this type. Here it may be mentioned that some authors (Hecht 1913 and Sbham 1929) have found in children more often the supraventricular than the ventricular type.

Campbell 1929 again, states that the nodal type is the more usual, but his observations are based upon a relatively small amount of material.

In judging the possible predisposing factors in the origin of the extrasystoles it appears that 9 of the children who showed the supraventricular type, had had different infectious diseases. The corresponding figure for children with the ventricular type was 5. It is possible that the origin of the extrasystoles in these cases may be interpreted as a result of a local stimulus due to infection within a limited area of the heart's specific musculature. As infectious diseases seem to occur relatively more often in connection with the supraventricular than with the ventricular extrasystoles the former may possibly be interpreted as a more serious symptom than the latter. One fact supporting this assumption is the observation that children with the supraventricular type showed relatively more often pathological symptoms of the heart than the cases where the extrasystoles had proceeded from the ventricle.

However, the examinations indicated that in most cases no possible predisposing factors could be found for the origin of the extrasystoles. Thus this should support the assumption that in children the extrasystoles are often »constitutionally» conditioned. Neurolability as a probable predisposing factor occurred in 17 cases (8 of them with supraventricular extrasystoles). This number is considerably higher than in the corresponding investigations of Lyon and Rauh; their material comprised 79 children with extrasystoles and they found neurolability in only 2 cases.

Of the children with supraventricular extrasystoles 12 had had subjective troubles such as dyspnoea, stitch in the region of the heart and fatigue. The corresponding figure for the ventricular type was only 5.

As regards the blood pressure and pulse frequency no deviation from normal conditions could be observed. Therefore it seems as if these factors play no important role in the origin of extrasystoles in children.

Observations regarding the electrical axis of the heart show that there is no apparent support for the assumption that a special preponderance of it is a deciding factor in the pathogenesis of the

extrasystoles. Thus, as regards the normal beats, in 73 cases there was no relative preponderance in the position of the electrical axis. Eleven children showed a right axis deviation and 2 a left axis deviation. On the other hand it is remarkable that in 31 cases the ventricular extrasystoles were of the right type and in only 11 cases of the left. This means, in other words, that the ectopic stimulus formation centre in cases of ventricular extrasystoles — according to the classical nomenclature — seem to be most often localised to the right ventricle.

Allorhythmias — bigeminy and trigeminy — occurred in 12 cases (2 of them of supraventricular origin). No clinical difference between these and other forms could be observed.

Extrasystoles of multifocal origin occurred in only 3 children. According to the observations made by D'IRSAY (1927) on adults such extrasystoles imply a worse prognosis than the unifocal forms. This could not be confirmed in the observations mentioned above.

Control examinations were carried out in 32 cases and showed the prognosis as to the health to be good in all cases. In 12 of the children extrasystoles were registered only at the first examination. These investigations showed that in those cases where the extrasystoles remained, the localisation of the ectopic stimulus formation was in the main unchanged. This indicates that the origin of the extrasystole in the single case is most often to be sought in disturbances within a limited area of the specific muscular system. Different infectious diseases seem to cause such disturbances. It is, however, probable that besides this matter a constitutional factor in the form of an abnormally strong stimulus formation capacity within a limited part of the specific muscular system is also to be considered. As a releasing element of the extrasystoles purely humoral factors must possibly also be given a certain importance. VESA (1939) has for example seen extrasystoles arise in adults after the injection of adrenalin.

The above results can be summarised under the following points: Extrasystoles are generally harmless disturbances in the heart's action and they occur not seldom in clinically healthy children. The ventricular extrasystoles are more usual than the supraventricular. The extrasystoles

seem to be relatively more usual in children with »myocardial damage» or heart disease than in healthy children. The extrasystoles are relatively often associated with neurolability. Clinical symptoms seem to occur more often in the supraventricular than in the ventricular form. On the other hand there seems to be no corresponding difference between the varying types of ventricular extrasystoles. The extrasystoles have a good prognosis.

Flutter and fibrillation

The auricular flutter and fibrillation are both characterised by an abnormally rapid auricular action. According to SHERF and BOYD the term auricular flutter is used in cases of rapid, co-ordinated auricular action marked by distinct, regular P-waves in the electrocardiogram and a partial atrio-ventricular block. The auricular fibrillation, on the other hand, indicates the condition in which the auricular action is completely irregular without any distinct registrable P-waves. This disorder is in addition usually characterised by a complete ventricular arrhythmia — delirium cordis. The transition from flutter to fibrillation is not distinct. Transitional forms between the two conditions is generally called fibrillo-flutter.

The detailed mechanism of the origin of auricular flutter and fibrillation is unknown. According to ENGELMANN (1896), HERING (1900) and WINTERBERG (1926) these forms of arrhythmia arise as a consequence of a high frequency stimulus formation in one or several ectopic centres in the musculature of the auricle. MAYER (1908), MINES (1914), GARREY (1914), and LEWIS (1920), on the other hand, who launched the so-called circus movement theory, consider that the stimulus formation in the auricular flutter passes over the auricular musculature in circular, regular waves. From this ring the stimulus impulses radiate out to the surrounding heart musculature in such a way that a contraction of the auricle corresponds to each completed circulation. In consequence of the rapid action of the auricle the ventricles are unable to respond to every impulse coming from the auricle and this expresses itself in varying

degrees of block. These authors also consider that a circus movement is the basis of the auricular fibrillation, but in this case the movement of the stimulus formation waves is irregular.

The difference between flutter and paroxysmal tachycardia has been the subject of discussion. According to one set of opinions no sharp distinction can be made between these two disorders (HUBBARD, 1941, SIEGEL, 1945, SHERF and BOYD, 1945).

In adult heart patients flutter and fibrillation are quite usual. SEMERAU (1921), MCEACHERN and BAKER (1932) as well as SHERF and BOYD (1945) estimate that flutter arrhythmias occur in about 50 % of all patients with heart decompensation.

In the etiology of these forms of arrhythmia the greatest importance is attributed to the following factors: arterio-sclerosis, different valvular diseases, hypertension and thyreotoxicosis. It is thus in the nature of things that flutter and fibrillation are relatively rare in children. COOKSON (1929) in 1164 hospital cases of auricular flutter found only 30 patients under 17 years of age. Perhaps even this figure is too large, as only 10 of those 30 were diagnosed electrocardiographically. In all the cases the fibrillation occurred in association with rheumatic infection. Signs of decompensation were observed in 22 of the children. Mortality was said to be about 50 %. The material of MCEACHERN and BAKER comprised 575 registered cases of auricular flutter and showed only 0.5 % of patients under 10 and 4.2 % between 10 and 20 years. SALTZMAN (1937) describes this kind of arrhythmia in 210 patients of whom 3.9 % were under 19.

The first cases of auricular flutter in children are said to have been published by NOUBÉCOURT and PICHON (1925) and by RESNIK and SCOTT (1926). They describe this arrhythmia in association with rheumatic infection. LEYS and RUSSELL (1927) describe a case of persistent auricular flutter in a 6-year-old boy with suspected diphtheria.

In contrast to earlier authors SCHWARZ and WEISS (1928) consider that auricular fibrillation is not quite unusual in children especially those with rheumatic infection. Thus they examined 16 children with rheumatic heart disease in the ages from 5 to 15 years for a period of 5 years, and auricular fibrillation appeared in

10 cases. The prognosis was bad because 7 of the patients died in the course of 1 year after the fibrillation was diagnosed. SCHMITZ (1932) in examining 1345 children under 15 years with heart disease found auricular fibrillation in 19 of them, the youngest being 8 years old. Of these 19 children 7 died on an average 1 ½ years after the appearance of the fibrillation. Since then similar observations have been made by ARANA and KREUTZER (1937) and by GIBSON (1941). The former examined 636 children with suspected heart disease and found among them 8 cases of auricular fibrillation, most of them having a fatal result. GIBSON reports 21 cases of auricular fibrillation obtained from 864 children with rheumatic heart disease. In 3 of them it was probably a result of digitalis medication. Congestive heart failure occurred in 18 cases; 14 of the children died.

GOLDBLOOM and SEGALL (1938) describe the first case of auricular fibrillation in a child under 1 year. It was a 3-months-old girl and she was cured. A similar case was later published by GAÁL (1944).

According to one prevalent opinion auricular flutter is considerably rarer in children than auricular fibrillation. Thus HALBERTSMA and HARTOG (1939), when reporting this form of arrhythmia in a 2-year-old girl with pneumonia, stated that they knew of only 3 cases of auricular flutter in children having been described earlier (AMBERG and WILLIUS, 1926, CARR and MCCLURE, 1931 and SHERMAN and SCHLESS, 1934). In studying the literature a few more cases have come to my knowledge. POYNTON and WYLLIS (1926) report a case of auricular flutter in a 4-months-old boy, whose heart action was normal when examined 3 ½ months later. This form of arrhythmia has also been observed by O'DONOVAN (1927) and by WEGMAN and EGBERT (1935). The former's case was a 5-year-old boy who had had diphtheria. Post-diphtheric auricular flutter in children has since been described by BEER (1937) and by BOLT (1940). In ARANA and KREUTZER's material comprising 636 children with heart disease there are 2 cases of auricular flutter. Further MANNHEIMER (1940) and HEDBERG (1945) have each described in detail a case of congenital flutter, the first with a fatal result. Finally NÁDRAI (1941) has seen this disorder in a child in associa-

tion with formalin poisoning. Thus the published number of cases of auricular flutter in children is at present only about 10.

Ventricular flutter and fibrillation is generally considered extremely rare in childhood. The lack of information in regard to these forms of arrhythmia is probably due to the fact that they usually shortly end in death and thus most often are not diagnosed. JOSEPHTAL (1934) has described a case of ventricular flutter in connection with diphtheria in a 4-year-old girl. She recovered.

A total of 6 cases of auricular flutter (0.11 %) was found in the material. In one patient the flutter temporarily became fibrillation under digitalis medication. No other case of fibrillation was found (2 premature infants showed temporary changes in the electrocardiogram resembling auricular fibrillation which however were interpreted as artefacts (see page 89).

The results are collected in table 9. In cases nos. 1 and 2, of which the latter was published by MANNHEIMER (1940), the flutter was congenital because tachycardia and insufficiency symptoms had existed since the birth. It is possible to imagine that the flutter in case no. 3 was also congenital, considering the relatively young age of the patient (1 year 11 months), although a possible connection between the arrhythmia and the acute pharyngeal infection cannot be excluded. Besides this case another one (no. 4) had an infectious disease (pulmonary tuberculosis) when examined. In the other cases there was no information as regards diseases which might have played a part in causing the flutter. One of the children (no. 4) had, for 2 months before the appearance of the flutter, been given digitalis on different occasions. In the other cases no medicine had been given before the flutter was diagnosed. None of the patients showed any sign of neurolability.

A bad general condition and signs of decompensation — cyanosis, dyspnoea and liver enlargement — were observed in 4 cases. In 5 of the children the heart was considerably enlarged, having a globular shape when examined radiologically.

Phonocardiograms were taken in 4 of the cases, and 2 of them showed an organically conditioned systolic murmur with a fre-

TABLE 9.

Auricular flutter.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Signs of decompensation	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram				Observation time	Course	
									Auricular frequency (beats per minute)	Ventricular frequency (beats per minute)	Block	»Myocardial damage«		Flutter	Prognosis
1	Girl	2 mths	—	Dyspnoea and cyanosis since birth	Bad	+	+	50—400	300	150	2: 1	+	1 wk	Remains	Death at 6 mths
2	Boy	3 ½ mths	—	Dyspnoea and cyanosis since birth	Bad	+	+	— (Auscultation)	400	200	2: 1	+	3 mths	Disappeared	Death at 7 mths
3	Boy	1 yr 11 mths	—	Acute tonsilitis	Unaffected	—	—	50—250	300—450	150	2: 1; 3: 1	+	1 yr 4 mths	Disappeared	Recovered
4	Girl	12 yrs	—	Active pulmonary t.b. Pancarditis	Bad	+	+	Systolic murmur (auscultation)	350	110	3: 1	+	7 mths	Disappeared	Convalescent
5	Boy	13 yrs 8 mths	—	Dyspnoea, Cyanosis	Bad	+	+	50—500	300	75	4: 1	+	1 yr 1 mth	Remains	Convalescent
6	Girl	15 yrs 6 mths	—	Systolic murmur	Unaffected	—	+	50—250	300	60—120	2: 1; 3: 1	—	2 mths	Remains	Free from discomfort

quency up to 400—500 cycles per second. In case no. 4 a strong systolic murmur was heard at the auscultation.

At the time of the diagnosis the auricular frequency varied in the different cases between 300 and 450, and the ventricular frequency between 60 and 200 beats per minute. The block type 2: 1 was the most usual and appeared in 4 of the cases. The highest degree of block, 5: 1, varying with 2: 1, was observed in case no. 6. All the children showed signs of »myocardial damage» on the electrocardiogram, such as pathologically formed initial deflections, changes in the S—T segments and negative or iso-electrical T-waves in at least two leads.

The period of observation varied in the different cases between 1 week and 1 year 4 months. They have all been followed with frequent controls. In 3 children (nos. 2, 3 and 4) the fibrillation disappeared after 3 days, 1 ½ months and 2 months respectively, while in the others the arrhythmia remained for the whole period. Two of the children, nos. 1 and 2, died at the ages of 6 and 7 months respectively. The former was treated at the hospital for only 1 week and discharged in a bad state at the parents' request. The latter child had also symptoms of decompensation when discharged, but the flutter had disappeared. While at the hospital an increased conduction time and the dropping of some ventricular complexes — probably an effect of digitalis — were observed on one occasion after the disappearance of the flutter.

In case no. 3 (Fig. 8 a—d), the flutter remained unchanged for 1 ½ months. Before it disappeared it became an auricular fibrillation with complete ventricular arrhythmia after digitalis medication. Afterwards the child was examined 11 times in all in the course of a full year and this revealed, with the exception of a temporary prolongation of the conduction time, no pathological signs.

One of the children (no. 3) became quite well, while another (no. 6) was quite free of trouble at the end of the observation period although the flutter still remained. In both these cases and in no. 2 the signs of »myocardial damage» in the electrocardiogram had disappeared at the last examination, while in the other cases these symptoms remained during the period of observation.

In all cases digitalis was used. In case no. 2 it had no effect

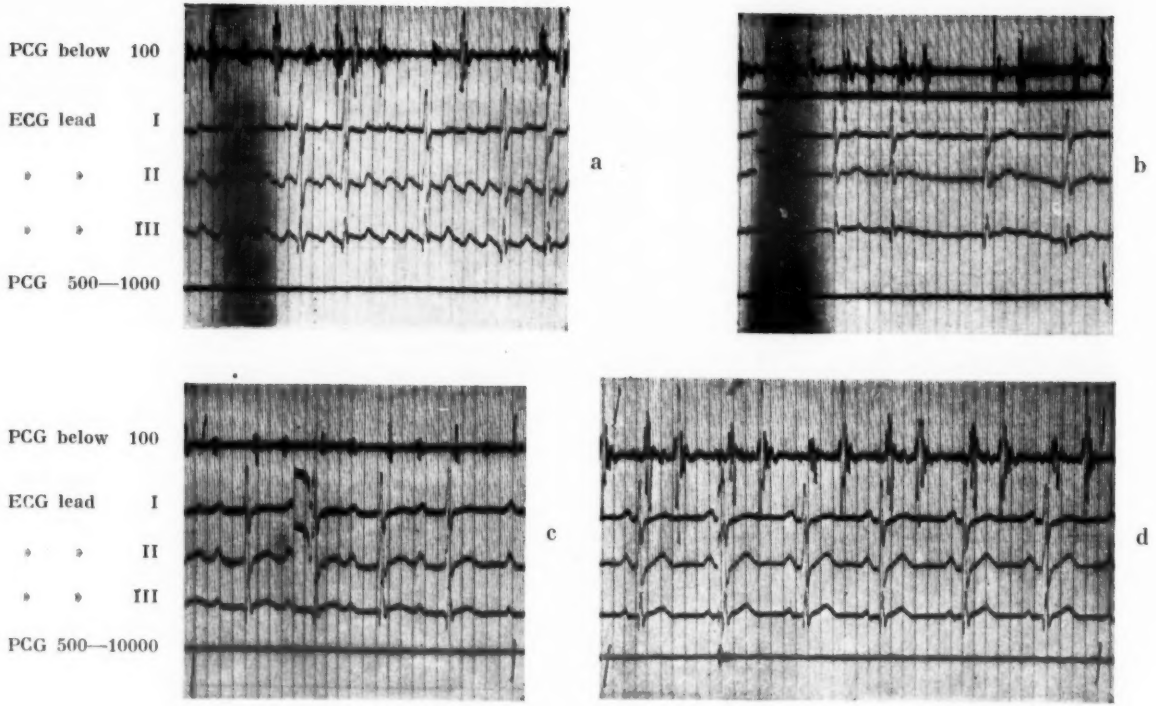


Fig. 8. Congenital auricular flutter in a boy of 1 year and 11 month (case no. 3).

a. 20. VI. 44. Auricular flutter with auriculo-ventricular block 2: 1, 3: 1.
b. 2. VIII. 44. After digitalis treatment the flutter is transformed into auricularfibrillation. Complete ventricular arrhythmia.
c. 3. VIII. 44. Continued digitalis treatment. Partial auriculo-ventricular block. P—Q interval of 0.26 second.
d. 15. VIII. 44. Normalised heart action. Sinus rhythm of about 90 beats per minute. P—Q interval of 0.15 second.

but after the administration of chinidin the flutter disappeared in 3 days. In case no. 4 chinidin was also used but without affecting the flutter.

Discussion. The results confirm the opinion that auricular flutter is a serious condition in children. Thus 2 of the 6 cases died in a relatively short time and most of the patients showed symptoms of decompensation when the flutter was diagnosed. In all cases signs of »myocardial damage» were also found in the electrocardiogram.

In attempting to form an idea of the etiology of the flutter on the basis of the observations made it appeared that the arrhythmia was congenital in 2 children. One can possibly assume that in these cases the flutter is due to a congenital injury which acts as a restraint on the normal stimulus formation. This assumption harmonises with the observation that in cases of flutter and fibrillation degenerative changes have been found in the sinus node (according to WENCKEBACH and WINTERBERG). In the other cases it is also hypothetically plausible that the disturbances in the stimulus formation have arisen on an anatomical basis. Perhaps the symptom of »myocardial damage» in all the electrocardiograms reflects an infection focus which has injured the normal mechanism of the stimulus formation. Another circumstance which favours the assumption of such an anatomical basis is that 3 of the patients showed an organically conditioned murmur. But the fact that the flutter disappeared in half of the cases shows that the disturbances of the stimulus formation are often not permanent. In this connection an opinion put forward by SALTZMAN (1937) can be mentioned according to which, in working out the etiology of the flutter and the fibrillation, an endogenous disposing factor should be considered besides those which are purely anatomically conditioned.

In 1 case the flutter became temporarily an auricular fibrillation after digitalis medication, a not unusual phenomenon in adults. It is remarkable that the material showed no other cases of fibrillation and this fact is in opposition to the very general opinion that auricular fibrillation is more common in children than auricular flutter.

c. 3. VIII. 41. Continued digitalis treatment. Partial auriculo-ventricular block. P—Q interval of 0.26 second.
d. 15. VIII. 41. Normalised heart action. Sinus rhythm of about 90 beats per minute. P—Q interval of 0.15 second.

In 2 children the auricular flutter probably disappeared in consequence of digitalis medication. A similar favourable effect of chinidin was noted in 1 case.

The above observations can be summarised as follows:

The auricular flutter is a relatively rare disorder in children and appears most frequently in association with other pathological changes in the heart. As a rule the clinical picture is dominated by definite decompensation symptoms. The prognosis is bad especially in cases of congenital flutter. Digitalis and chinidin have at times a favourable therapeutic effect. Auricular fibrillation seems to be relatively rarer than auricular flutter.

Paroxysmal tachycardia

Paroxysmal tachycardia is characterised by a sudden abnormal increase of the heart frequency. It is distinguished from the other tachycardias by the sudden beginning and equally sudden ending of the attack.

Lewis (1904) was one of the first to distinguish a supraventricular and a ventricular type of paroxysmal tachycardia according to the localisation of the ectopic focus which dominates the heart action. It has been thought that this disorder arises as a result of a rapid rhythmic stimulus formation in such an ectopic centre and it can thus be compared to a long chain of multiple extrasystoles of either supraventricular or ventricular origin. It has already been stated that the transition between the paroxysmal tachycardia and flutter is not distinct. In interpreting an electrocardiogram it can therefore sometimes be difficult to decide whether it shows a paroxysmal tachycardia or an auricular flutter of the type 1: 1 (Hubbard 1941).

Published cases of paroxysmal tachycardia in children are relatively few. The first collocation was made by Taran and Jennings (1937) and includes 52 cases in children under 15 years. To this number they added one observation of this disorder in a new-born infant. In 1940 Johnson mentioned, in reporting a case of paroxysmal tachycardia in an 11-year-old boy, that after the account of Taran and Jennings a further 11 cases of this disorder in children had been described. Further such cases have been published by Keith and Brown (1940), Cammarella (1941), Hobbs (1941), Hubbard (1941), Rosenbaum, Johnston and

KELLER (1942), LEBRET and ERKELENS (1942), also by CHRISTENSEN (1944), DÜNZELMANN (1944), HOWARD (1945), and finally by FRISELL (1946) and by MANNHEIMER (1946). HUBBARD's and HOWARD's material is the most extensive and comprises 9 and 6 children respectively. The observations of the others only concern solitary cases.

Thus at the present time the published cases of paroxysmal tachycardia in children amount to about a hundred. According to CHRISTENSEN's summary 37 of them were under 1 year. This number is increased to 43 by the cases of HOWARD, FRISELL and MANNHEIMER, which show that the disturbance in children appears relatively often during the earliest period of life.

Of the two types the supraventricular form seems to be the most usual. Thus ROSENBAUM, JOHNSTON and KELLER (1942) counted only 26 published cases of ventricular paroxysmal tachycardia in children and added to these 2 of their own cases. Paroxysmal tachycardia of nodal origin is extremely rare in childhood. TARAS and JENNINGS (1937) mention 5 published cases (one of their own). Later WRIGHT (1938), and PUGLISI (1939) have each described a case of this form of paroxysmal tachycardia in children.

While the symptoms in older children do not specially differ from those found in adults, this disorder in infants usually presents clinical pictures differing in many respects. As these differences have been explained only lately — chiefly perhaps thanks to HUBBARD's investigations — it is almost possible to describe paroxysmal tachycardia in infants as a relatively new disease in pediatrics. The attack in infants has often non-characteristic initial symptoms such as anorexia, vomiting and listlessness. Gradually, however, insufficiency symptoms arise with cyanosis, dyspnoea, liver enlargement, pulmonary congestion, and a general condition often resembling collapse. The attack lasts as a rule some hours but can in rare cases last several days. At the offset the clinical symptoms disappear in a short time.

Even if the prognosis as regards health in paroxysmal tachycardia in infancy is often good the attacks cause some anxiety for the child's future on account of their relatively great tendency to recur. Experience has shown the importance of therapeutic

measures as early as possible. Different authors have traced a favourable effect to digitalis (Farr and Wegman 1935, Taran and Jennings 1937, Püschkel 1939, Hobbs 1941, Hubbard 1941, Howard 1945 and Frisell 1946). Thus Hubbard cut the attack in 8 of his 9 reported cases with this drug. Yet digitalis treatment can cause certain complications such as complete block (Howard 1945). Christensen has observed a good effect from strophantin. Some other authors attribute importance to mecholyl — an acetylcholin derivate. — A favourable effect of such derivates is reported by Wright (1938) and by Walsh and Sprague (1940). One disadvantage is, however, their rather pronounced toxic effects such as vomiting and diarrhoea. Recently Mannheimer (1946) has observed a good effect of neostigmin (=prostigmin). In both his cases the attacks were cut with this preparation. Here it may be stated that Ask-Upmark (1941) has found an obvious decrease in the heart frequency of healthy adults after administering neostigmin. In contrast to the conditions in older children and adults carotid- and bulbar pressure has most frequently no therapeutic effect on paroxysmal tachycardia in infants.

The opinion is generally held that the prognosis chiefly depends upon the condition of the heart. Of the 43 published cases in infancy 7 ended fatally; 3 of them had congenital heart disease. Cases of death among older children have also been generally those with different heart diseases.

Paroxysmal tachycardia was found in 5 cases of the material (0.09 %). Of these cases, shown in Table 10, nos. 1 and 2 have been published by Mannheimer (1946).

No direct causative factors could be diagnosed in any of the children. One of them (no. 4) had previously had attacks of palpitation. In case no. 2 the tachycardia was diagnosed at the routine examination by the physician at an Infant Care Centre. The other patients fell ill suddenly.

Two of the patients, nos. 1 and 3, when admitted to hospital, showed a bad general condition and heart decompensation —

TABLE 10.

Paroxysmal tachycardia.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Insufficiency symptoms	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram				WPW-Syndrome	Total duration of attacks	Observation time	Course	
									During attack		After attack					Paroxysmal tachycardia	Prognosis
									Frequency (beats per minute)	»Myocardial damage«	Frequency (beats per minute)	»Myocardial damage«					
1	Boy	3 wks	—	Acute attack	Exhausted	+	+	50—250	300	+	145	—	+	2 days	6 mths	New attack after 1 mth	Recovered
2	Boy	3 wks	—	Tachycardia detected at Infant Care Centre	Unaffected	—	—	50—175	270	+	150	—	—	8 days	1 mths	No relapse	Recovered
3	Boy	2 yrs 4 mths	—	Acute attack	Exhausted	+	+	— (auscultation)	240	+	95	—	—	4 hours	2 wks	No relapse	Recovered
4	Girl	13 yrs 4 mths	—	Acute attack	Unaffected	—	+	50—250	225	+	80	+	+	3 hours	2 ½ mths	New attack after 1 mth	Recovered
5	Girl	13 yrs 9 mths	—	Acute attack	Unaffected	—	+	50—250	230	+	75	—	—	9 hours	1 yr	No relapse	Recovered

dyspnoea, cyanosis and liver enlargement. In the other cases the tachycardia was the only obvious symptom. Four of the children when examined röntgenologically showed a slightly enlarged heart.

The phonocardiographic examination, made in 4 cases, revealed normal sound phenomena. The fifth child had no murmurs at auscultation.

In all the children the paroxysmal tachycardia was of supraventricular origin. The heart frequency varied in the different cases between 225 and 300 beats per minute during an attack. When the tachycardia was diagnosed all the children showed signs of »myocardial damage» in the electrocardiogram, such as depressed S—T segments and negative or iso-electric T-waves in at least two leads. After the attack these changes remained in only 1 child (no. 4). The period of the attacks varied from some hours to 8 days, during which time several attacks generally occurred.

The patients' development has been followed with frequent controls for periods varying from 2 weeks to 1 year. Relapses occurred in 2 children (nos. 1 and 4), in both cases 1 month after the first attack. All the children recovered and showed no pathological symptoms at the latest examination.

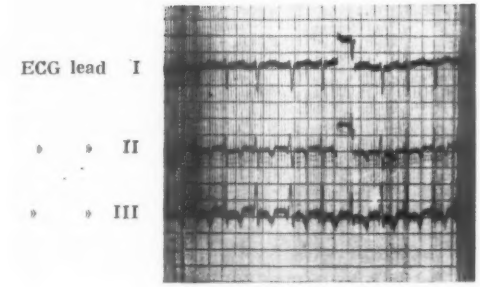
Lately several authors have drawn attention to the fact that patients suffering from paroxysmal tachycardia sometimes show signs of pre-excitation or the so-called WPW syndrome (Wolff, Parkinson and White 1930, Faxén 1936, Öhnell 1944, Jörgensen 1945, Lind 1945, Mannheimer 1946). The syndrome, of which the most prominent sign is a short P—Q interval combined with a cut-off on the R-waves' ascending part, has been interpreted as due to a reflex of the impulses from the ventricles, which causes an auricular contraction before the next impulse from the sinus node. Mannheimer points out that the ECG-values demanded by Öhnell and Christensen of pre-excitation, namely, a P—Q interval below 0.12 second and a broadening of the QRS complexes to over 0.10 second, are too high in the case of infants. According to Mannheimer, consequently, the criteria for small children must be used in accordance with the normal values for the age in question. The WPW-syndrome is unusual in children. Thus Jörgensen (1945) reports only 7 published cases.

In the above investigations signs of pre-excitation were seen in 2 children (nos. 1 and 4). The breadth of the QRS complexes was in these cases 0.12 and 0.11 second respectively. At the latest examinations the corresponding values were 0.04 and 0.05 second. Öhnell, like Mannheimer, has considered the possibilities of interpreting this decrease in some cases as an apparent return to normal conditions (pseudo-normalisation).

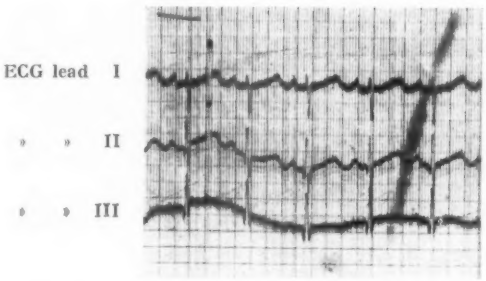
In treating the attack neostigmin (= prostigmin) was used with good effect in 2 cases, nos. 1 and 2 (fig. 9). In 1 child, (no. 4), the tachycardia changed into a normal sinus rhythm after bulbar pressure. In the 2 remaining children the heart action was normalised after treatment with fenemal and bromural. Carotid- and bulbar pressure had no effect in these cases.

Discussion. Several authors have lately held that paroxysmal tachycardia is probably considerably more usual in children than the published cases show (Hubbard 1938, Christensen 1945 and Mannheimer 1946). This assumption refers chiefly to infants in whom the secondary symptoms in an advanced stage of an attack can easily be misinterpreted because the tachycardia at the basis of the symptoms has been overlooked. In such cases the clinical picture can often be confused with, for example, pneumonia or with other diseases accompanying a low general condition. Thus the possibility of a faulty diagnosis is relatively great. One of Christensen's cases, for example, was sent by a doctor to the hospital with suspected invagination. As, in addition, paroxysmal tachycardia can sometimes cease spontaneously in infants, giving rise only to the non-characteristic initial symptoms, the assumption is justifiable that in such cases the disease is often not diagnosed. Further it should be noted that the discovery of this disorder is very much more difficult in children because of the relatively pronounced tachycardia normal at this age.

In the above cases the typical insufficiency symptoms were observed in 2 children, 3 weeks and 2 years 4 months old respectively (cases 1 and 3). Similar symptoms were lacking in the other infant (case 2), probably because they had not yet developed as the different attacks could be cut at an early stage. Hubbard reports a similar case in which paroxysmal tachycardia was discovered



a



b

Fig. 9. Paroxysmal tachycardia in a boy of 3 weeks (case no. 2).

a. During attack. Heart frequency 270 beats per minute.

b. Five hours later, after injection of 1/8 mg neostigmin. Normalised heart action. Heart frequency 150 beats per minute.

in an infant at the routine examination when the patient was about to be discharged from hospital.

The investigations confirm a rather general experience that it is often difficult to find any direct predisposing factors in the origin of this disturbance in children. Thus in none of the 5 cases did the attack arise in connection with an infectious disease, nor did any of the children show signs of organic heart disease. Theoretically it can thought that the tachycardia is partly rendered possible by some process, perhaps infectious, which has disturbed the mechanism of the normal stimulus formation. Here it can be stated that some of the authors already mentioned have seen paroxysmal tachycardia arise in connection with infectious diseases such as rheumatic fever, pneumonia and diphtheria. Speaking of etiological factors HUBBARD mentions intra-cranial injuries. v. BERNUT and v. DEN STEINEN 1930 have observed paroxysmal tachycardia in encephalitis. In the numerous cases where it has not been possible to find any direct causative factors, some authors — among them CHRISTENSEN — consider that the tachycardia is a consequence of a failure in the central or autonomous regulation of the heart. That the disease is rather often found in infants is, according to him, due to the fact that different regulation mechanisms are not yet fully developed at this stage of life.

It has already been mentioned that the transfer between paroxysmal tachycardia and the other active heterotopias is sometimes not distinct. It is therefore not impossible that a similar mechanism of origin is at the basis of these different forms of arrhythmia. In that case the difference between them would be rather quantitative than qualitative.

That all the 5 children recovered can probably be attributed to the fact that none of them showed signs of any serious disease The prognosis as to the health is, however, not quite favourable in that in 2 children the disease recurred. Here it may be stated that DÜNZELMANN (1944) followed the development of 6 children who had had attacks of paroxysmal tachycardia and noticed relapse in 4 of them.

Summary: *Paroxysmal tachycardia is rare in children but relatively more usual in infants. It is often difficult to find a direct cau-*

sative factor for this disturbance. The clinical picture is essentially different in infants from that found in older children and adults. In infants it is easily overlooked because of its non-characteristic initial symptoms. During the attack the picture at this age is often dominated by distinct insufficiency symptoms which can give rise to misinterpretation. Apart from the rather great risk of relapse the prognosis is generally good if there is no other more serious form of heart disease. In treating the illness it seems that, besides digitalis and strophantin, derivates of acetylcholin and above all neostigmin or prostigmin are valuable medication. [1]

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[1] Since my return to Helsingfors I have had the opportunity of observing 4 cases of paroxysmal tachycardia in infants at the University Children's Clinic. Two of them were new-born and they showed typical signs of decompensation. The one died shortly after arriving at the hospital, the other recovered. The latter was given neostigmin. The remaining cases were two 3-month-old infants in whom the attacks were also cut with neostigmin. No signs of decompensation could be found and the children recovered.

Conduction disturbances

The stimulus impulses are led from the sinus node, the heart's normal pacemaker, in the form of rays over the auricles. From the right auricle the conduction passes via Tawara's node to the bundle of His, and thence through both its branches and their divisions to the musculature of the ventricle.

Disturbances in the conduction can arise in all sections of the specific muscular system. According to their intensity different degrees of block are distinguished. The slightest form is marked only by a retardation of the conduction in a limited part of the heart and does not cause any disturbance in the heart rhythm. On the other hand, if the obstacle is greater it can cause a dropping of solitary contractions in the caudal parts of the heart and an arrhythmia will result (a block of the second degree). In a block of the third degree the conduction to the ventricles is completely interrupted. In this condition the action of the auricle is completely independent of the ventricles, which beat in their own rhythm. Because of this dissociation between the two rhythms the complete block can be referred to the arrhythmias, although both the auricle and the ventricle are beating regularly. It is characteristic of this condition that the rhythm then arising is the slower the more caudally the interruption is situated.

Disturbances in the conduction can arise in different ways. In childhood the block caused by congenital malformations in the conduction system has a special position. Another main group is

formed of conduction disturbances of an infectious-toxic origin. The retarded conduction, for example in a myocarditis, is quite generally considered to be due to nutrition disturbances in the heart characterised by a collecting of toxic decomposition products with bad conduction ability in a limited section of the specific muscular system (v. KISS, 1936, HOLTZMANN 1945). According to some authors the cause of the decreased conduction ability in such cases could also be attributed to an increased vagal tonus (KEITH 1938). This assumption is supported for example by the observation that a supply of atropin increases the heart's conduction ability (HECHT 1912). Further, in childhood factors of less importance may cause a decrease in the conduction ability. To these belong e.g. the purely functional disturbances and blocks caused by different drugs.

Sino-auricular conduction disturbances. Sino-auricular blocks are rare because of the rich occurrence of conduction elements between the sinus node and the auricle. A partial block in this section results in a slow but regular pulse. The consequence of a pronounced bradycardia can, however, be that an ectopic centre, because of the decreased control from the sinus node, becomes active and gives rise to contractions, so-called escaped beats, which break the regular rhythm (passive heterotopy). Usually such beats proceed from Tawara's node which within the specific muscle system works with the highest frequency intensity next to the sinus node. As distinct from the extrasystoles the previous pulse interval is longer than between the normal beats. I have been unable to find in the literature any more detailed information regarding the occurrence of escaped beats in children. MIKULOWSKI (1936) has described this form of arrhythmia in an 8-year-old girl with congenital syphilis. In general it is not considered very important clinically. According to SHERF and BOYD escaped beats are rather the expression of a deliberate protective mechanism in the heart.

Sino-auricular block of the second degree is characterised by an intermittent drop of one or several heart contractions. Electrocardiographically the block is recognised because both the auricular and the ventricular contractions are absent during the

pauses. Published cases of sino-auricular blocks of the second degree in children are very few. WICKSTRÖM (1933) has observed this form of arrhythmia in connection with scarlatina. v. KISS (1936) has described sino-auricular block in a 5-year-old girl with diphtheria. KRISTZ (1937) has published a case of sino-auricular block in a new-born child. According to NÁDRAI (1941) this form of arrhythmia is not quite unusual in different infectious diseases, such as scarlatina, diphtheria, typhus and rheumatic fever.

When the sino-auricular block is complete the control of the heart action is usually taken over by Tawara's node, though under these conditions the heart beats in the regular nodal rhythm without causing any arrhythmia in the real sense.

Atrio-ventricular block of the second degree is distinguished by the fact that the stimulus impulses are intermittently not transferred from the auricles to the ventricles because of some obstacle. The intensity of this obstacle allows the differentiation of two forms of interruptions, types I and II. In the former the stimulus impulses are delayed from beat to beat, which is reflected in the electrocardiogram by a successive increase of the P—Q interval. The consequence of this cumulative exhausting of the conduction is that in the end the impulses are not transferred, and a ventricular contraction is dropped. After this absence the P—Q interval is shortest and then increases again until the next dropping of the ventricular contraction (WENCKEBACH-arrhythmia).

An atrio-ventricular block of type II is marked by a dropping of the ventricular contraction without a previous prolongation of the P—Q interval. The degrees of blocking are distinguished according to the intensity of the interruption, such as 2: 1, 3: 1 and 4: 1 blocks.

While a simple prolongated conduction time is a relatively common occurrence in infectious diseases — WICKSTRÖM for example, reports a frequency of 67 % in scarlatina — an atrio-ventricular block of the second degree is rare in childhood. Thus LEMANN (1933) in an electrocardiographic examination of 3000 people under 30 years of age found this type of block in only 6 cases.

Of the two types of atrio-ventricular block of the second degree

it seems that the WENCKEBACH arrhythmia is relatively the most usual. WICKSTRÖM (1935) and BEER (1938) describe 2 and 3 cases respectively of this form of arrhythmia in children in connection with scarlatina. Later v. KISS and VOLTAY (1941) made similar observations. The case was a 5-year-old girl with a WENCKEBACH arrhythmia in connection with pneumonia. NÁDRAI (1941) in examining 80 children suffering from typhus found this form of arrhythmia in 1 case.

An atrio-ventricular block of type II seems also especially to appear in connection with different infectious diseases such as rheumatic fever and pneumonia, according to the observations of v. KISS and WOLLEK (1935), GUPTA (1937) and NÁDRAI (1941).

Among the complete atrio-ventricular blocks the congenital obstacles in the conduction are of great interest because of what is considered the mechanism of their origin. YATER (1929) has set up the following criteria for them:

1. The block shall be diagnosed electrocardiographically in relatively young individuals.
2. Bradycardia must have occurred at an early age.
3. To exclude the possibility of the block having been contracted there must be no infectious diseases — specially diphtheria and rheumatic fever — in the case history.

Proceeding from these criteria YATER, LYON and MCNABB in 1933 collected 44 cases of congenital block in children described up to that time. To this number they added 3 of their own observations. Detailed comparisons of congenital blocks have later been given by NIELSEN (1935) and by WALLGREN and WINBLAD (1938). The former reported 2 of his own cases, one of which was published by GJØRUP (1933). WALLGREN and WINBLAD increased the number of published cases to 77 by a detailed description of congenital block in two infants. Since then further cases have been observed (CURRIE 1940, GEIGER and LAURENCE 1940, GORTER 1940, MANNHEIMER 1940, WISSLER 1940, NÁDRAI 1941, KÜHN 1941, PEEL 1943, SJÖQVIST 1943, THOMSON 1943, HEDBERG 1944, HOEKENGA 1945). The number of published cases at the present time is thus about 100.

Our conception of the more detailed mechanism of the origin of the congenital block is still relatively incomplete. Yet one is most inclined to make a septum defect responsible for the interruption in the conduction system. Besides such a mechanism it is possibly also necessary to reckon with a fetal inflammatory process creating a obstacle in the conduction (WALLGREN and WINBLAD). Certain facts are specially in favour of the former assumption. Thus 66 % of the children in WALLGREN's and WINBLAD's report showed clinical symptoms of a septum defect. A great number of these children, however, also showed signs of other malformations in the heart, such as pulmonary stenosis and patent ductus Botalli. According to WISSLER 11 cases had been examined post-mortem up to 1940. Among these septum defect was found in 4 children, while the rest showed no obvious pathological-anatomical changes in the heart. Of the cases of congenital heart block in children published lately HOEKANGA's and PEEL's showed a septum defect at the post-mortem.

According to information in the literature mortality among children with congenital block is about 25 %. Most of the deaths have occurred in the first year of life. Where there is strong cyanosis the prognosis is specially bad. NIELSEN reports a mortality of ca 80 % among such children. On the other hand if there are no serious changes in the heart, it seems that the block in itself does not cause the patient any direct trouble. KÜHN (1942), for example, describes a case of persistent congenital block in a newborn infant who developed normally until the latest examination at the age of two.

According to LEMANN's collection of 1933 about 60 % of complete heart block in children are contracted. The most important predisposing factors seem to be rheumatic infection, diphtheria and influenza. Post-diphtheric blocks have very often a bad prognosis. NÁDRAI (1941) mentioned 10 fatal cases among 12 children with complete block after this disease. It is, however, remarkable that contracted complete block can remain for a long time without causing the patient any direct trouble. In this connection it may be mentioned that v. KISS (1938) observed complete block after diphtheria in two children in whom the block remained for 5 and 10

years respectively. Bower (1939) has published the only cases known of persistent heart block in children after measles.

Information as to the treatment of atrio-ventricular block in children is very scarce. Some authors have stated that sympaticus stimulating drugs such as atropin and ephedrin can bring about a normalisation of the conduction system (Gupta 1937, Currie 1940 and Siegel 1945).

Escaped beats.

Arrhythmias in the form of escaped beats were found in 6 children in my material (0.11 %). The results of the investigation are collected in Table 11. In no case did the case history report previous infectious diseases. Three of the children were admitted to hospital because of an acute infectious disease (tonsilitis, peritonitis and polyarthritis). There was hypothyreosis in 1 case and orthostatic albuminuria in another. Four of the children showed signs of neurolability. The blood pressure varied between 90 and 120 mm Hg. The general condition was low only in the patient with peritonitis (no. 3). None of the children showed signs of decompensation.

In all cases a systolic murmur was registered phonocardiographically, but its frequency fell within physiological limits (up to 250 cycles per second). Heart enlargement was diagnosed only in the patient with polyarthritis, and this child was also the only one who showed signs of »myocardial damage» in the electrocardiogram (the P—Q interval was 0.24 second).

In all the cases there was a pronounced bradycardia as regards age varying between 50 and 65 beats per minute. In 5 children the passive heterotopic beats proceeded from Tawara's node, in 1 case the escaped beats originated in the ventricle (no. 4, Fig. 10). Case no. 5 was peculiar in that the escaped beats were noted only when the child was in a lying position. When the patient stood up the pulse rose to 90 beats per minute and the heart action became normal.

TABLE 11.

Escaped beats.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Signs of decompensation	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram			Course	
									Dominant rhythm (beats per minute)	»Myocardial damage«	Origin of escaped beats	Observation time	Escaped beats
1	Boy	4 yrs 4 mths	—	Acute tonsilitis	Unaffected	—	—	50—200	65	—	Tawara's node	1 mth	Disappeared
2	Girl	7 yrs 3 mths	—	Hypothyreosis. Enuresis noct.	Unaffected	—	—	50—250	50	—	Tawara's node	4 mth	Disappeared
3	Girl	9 yrs 1 mth	—	Acute peritonitis	Bad	—	—	50—250	60	—	Tawara's node	2 wks	Disappeared
4	Boy	10 yrs	—	Systolic murmur discovered by school doct.	Unaffected	—	—	50—250	55	—	Left ventricle	3 wks	Disappeared
5	Boy	12 yrs 8 mths	—	Orthostatic albuminuria	Unaffected	—	—	50—250	60	—	Tawara's node	3 wks	Disappeared
6	Boy	13 yrs 5 mths	—	Acute polyarthritis	Unaffected	—	+	50—250	60	+	Tawara's node	4½ mth	Disappeared

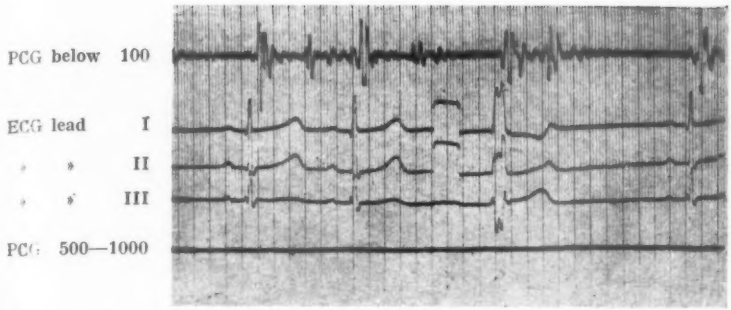


Fig. 10. Escaped beats of ventricular type in a girl of 10 years (case no. 4).

In all the cases the escaped beats disappeared without any treatment, mostly in the course of few days. The pulse frequency had then risen and varied in the different cases between 70 and 90 beats per minute. At the end of the examination period all the children were symptom-free.

Sino-auricular block.

The material included 7 cases of sino-auricular block (0.13 %).

From Table 12 it appears that the arrhythmia had in no case arisen in connection with an infectious disease. Four of the children (nos. 2, 4, 5, and 6) showed signs of neurolability. The general condition was good in all cases and there were no signs of decompensation. Heart enlargement was observed in 2 children (nos. 6 and 7). The pulse was normal as was the blood pressure which was measured in the 4 oldest children (the figures varied between 105 and 120 mm Hg). In 2 cases (nos. 5 and 7) there was a loud systolic murmur.

No other pathological changes besides the block could be observed electrocardiographically.

In one case (no. 5) the block disappeared immediately after the injection of bellafolin, in the others the rhythm became normal without any specific medication mostly in the course of some days. During the period of observation the children remained well.

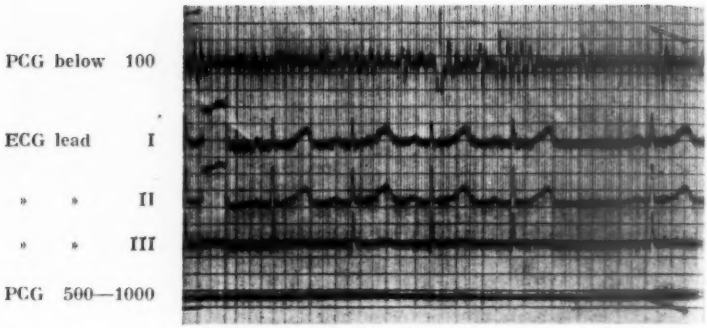


Fig. 11. Partial sino-auricular block in a girl of 5 years and 1 month (case no. 3).

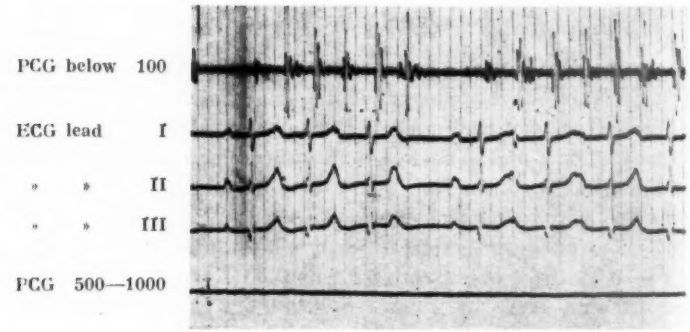
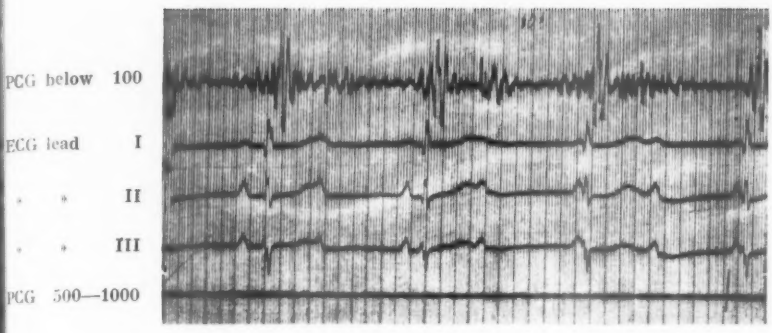
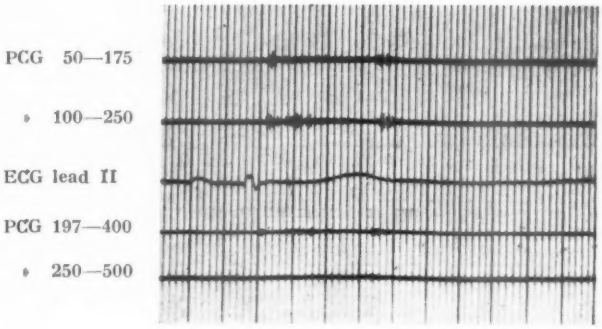


Fig. 12. Partial atrio-ventricular block type I — WENCKEBACH arrhythmia — in a boy of 5 years (case no. 1). Successive prolongation of P—Q interval resulting the dropping of each 4—6th beat.



a



b

Fig. 13. Congenital complete auriculo-ventricular block in a boy of 6 years (case no. 1, the latest control examination).

a. The electrocardiogram shows a total dissociation of the auricular and ventricular actions.

b. In the phonocardiogram a systolic murmur up to 400 cycles per second is registered.

TABLE 12.

Sino-auricular block.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Signs of decompensation	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram			Course	
									Dominant rhythm (beats per minute)	Dropped beats	»Myocardial damage«	Observation time	Block
1	Boy	2 mths	—	Arrhytmias since birth	Unaffected	—	—	50—250	110	Every 3—4th	—	2 mths	Disappeared
2	Girl	5 yrs	—	Anorhexia	Unaffected	—	—	50—250	90	Every 7th	—	1 wk	Disappeared
3	Girl	5 yrs 1 mth	—	Bronchitis (no fever)	Unaffected	—	—	50—250	100	Every 7—8th	—	1 ½ mths	Disappeared
4	Boy	7 yrs 3 mths	—	Debilitas mentis	Unaffected	—	—	50—175	105	Every 4—7th	—	1 wk	Disappeared
5	Girl	7 yrs 8 mths	—	Systolic murmur	Unaffected	—	—	50—1000	80	Every 3—4th	—	6 wks	Disappeared
6	Boy	9 yrs 10 mths	—	Debilitas mentis	Unaffected	—	+	50—250	70	Every 3—4th	—	1 ½ mths	Disappeared
7	Boy	11 yrs	—	Systolic murmur	Unaffected	—	+	moderate (auscultation)	100	Every 3rd	—	2 wks	Disappeared

Partial atrio-ventricular block.

In Table 13 are collected the cases of atrio-ventricular block of the second degree, 5 in number (0.09 %).

From this summary it appears that the arrhythmia in 4 of the children arose in connection with an active or earlier infectious disease. One of the children (no. 2) had been given digitalis before the appearance of the block. Acute polyarthritis was present in 2 cases (nos. 2 and 5) and in them the general condition was bad, in the others unaffected. All the children had enlarged hearts but showed no signs of decompensation. The blood pressure measured in the 3 oldest children was 115, 70 and 140 mm Hg respectively.

The phonocardiographic examination showed in 2 of the children (nos. 1 and 5) a probably organically conditioned murmur with a frequency up to 400—500 cycles per second.

The dominant rhythm varied in the different cases between 75 and 120 beats per minute. In 4 of the children the block was of the WENCKEBACH type. Dropped ventricular complexes without an increased P—Q interval occurred in 1 case (no. 5). In cases nos. 2, 4 and 5 the electrocardiogram showed, besides the block, other changes termed »myocardial damage» such as pathological initial deflexions, changes in the S—T segments, and negative or isoelectric T-waves in at least two leads.

The development of the cases was followed by frequent controls. In 3 of the children (nos. 2, 4 and 5) the block was registered only on one occasion. In 1 case (no. 3) the block disappeared in 2 days but reappeared temporarily on two occasions, 2 months and 2 years later respectively. In 1 child (no. 1) the block remained during an observation period of 9 months. All the children were in good health when last examined.

Complete atrio-ventricular block.

In going through the material a total of 9 cases of complete atrio-ventricular block (0.16 %) was found. Nos. 1, 4 and 8 of these were published by MANNHEIMER in 1940.

Table 14 shows that none of the children had had any infectious

TABLE 13.

Partial atrio-ventricular block.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Signs of decompensation	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram				Course	
									Dominant rhythm (beats per minute)	Block type	Dropped beats	»Myocardial damage«	Observation time	Block
1	Boy	5 yrs 6 mths	—	Anorhexia	Unaffected	—	+	50—400	100	I	Every 4—6th	—	9 mths	Remains
2	Girl	10 yrs 7 mths	—	Acute poly-arthritis	Bad	—	+	50—250	120	I	Every3 —5th	+	2 yrs	Disappeared
3	Girl	11 yrs 4 mths	Septic pancarditis	Systolic murmur	Unaffected	—	+	50—250	90	I	Every 5th	—	3 yrs 9 mths	Disappeared
4	Girl	12 yrs 8 mths	Scarlet fever	Repeated tonsilitis	Unaffected	—	+	50—250	75	I	Every 5—6th	+	1 mth	Disappeared
5	Boy	14 yrs 9 mths	—	Acute poly-arthritis	Bad	—	+	50—500	90	II	Every 2—4th	+	4 yrs	Disappeared

TABLE 14.

Complete atrio-ventricular block.

TABLE 14.

Complete atrio-ventricular block.

Case number	Sex	Age	Previous infectious diseases	Cause of admission to hospital	General condition	Signs of decompensation	Heart enlargement	Systolic murmur (cycles per second)	Electrocardiogram			Course	
									Auricular frequency (beats per minute)	Ventricular frequency (beats per minute)	»Myocardial damage	Observation time	Block
1	Boy	4 hrs	—	Bradycardia	Unaffected	—	—	50—400	100	45	—	6 yrs	Remains
2	Girl	1 ½ yrs	—	Systolic murmur	Unaffected	—	+	50—400	150	65	—	2 mths	Remains
3	Boy	1 ½ yrs	—	Bradycardia and systolic murmur	Unaffected	—	+	50—500	120	75	—	—	—
4	Girl	1 yr 10 mths	—	Systolic murmur	Unaffected	—	+	50—1000	100	50	—	4 ½ yrs	Remains
5	Girl	4 yrs 7 mths	—	Bradycardia and systolic murmur	Unaffected	—	+	50—500	105	50	—	8 mths	Remains
6	Girl	4 yrs 10 mths	—	Systolic murmur	Unaffected	—	+	50—500	95	55	—	1 ½ yrs	Remains
7	Girl	7 yrs	—	Systolic murmur	Unaffected	—	—	50—500	100	50	—	—	—
8	Boy	9 mths 3 yrs	—	Systolic murmur	Unaffected	—	+	50—400	70	45	—	2 ½ yrs	Remains
9	Girl	11 yrs 1 mth	—	Bradycardia and systolic murmur	Unaffected	—	—	50—400	80	45	—	5 yrs	Remains

diseases of clinical importance. In 1 case (no. 1) bradycardia was diagnosed by auscultation several days before birth, and in another (no. 3) immediately after it. Two other children (nos. 5 and 9) had been physically examined at the age of 2 and 3 years respectively, and then showed an abnormally slow heart action. The other children were sent to hospital to have a systolic murmur investigated.

All the children were in good general condition and there were no decompensation symptoms. The röntgenological examination revealed heart enlargement in 6 of them. The blood pressure — measured in the 7 oldest children — showed normal values varying between 80 and 135 mm Hg. The sedimentation rate was normal in all cases.

For all the children a strong systolic murmur with a frequency up to 400—1000 cycles per second was registered in the phonocardiogram.

When examined electrocardiographically a complete dissociation between the action of the auricle and the ventricle was diagnosed in every case. The auricular frequency varied in the different cases between 70 and 150 beats per minute, while the corresponding values for the ventricular frequency lay between 45 and 75 beats per minute. Except for the block no other definite pathological signs could be observed in the electrocardiograms.

Two of the children (nos. 3 and 7) were examined only once. In the other cases the development was followed by frequent controls during the period which varied between 2 months and 6 years. In all these children the block remained unaltered but no other signs of trouble could be observed and the children developed normally. No medicine was given. One child (no. 5) was narcotized in connection with an operation, without any heart complication.

Discussion. In judging conduction disturbances of the type called escaped beats it seems clear that the condition necessary for the rise of this form of arrhythmia is a slow heart action. This is well illustrated by observing that the arrhythmia disappeared in all 6 cases at the same time as the pulse conditions became normal. As an etiological factor in the origin of these beats, rheumatic fever seems to have played a certain part in 2 cases. Observation in this respect confirms the investigations of Edén (1940) who diagnosed

in children with acute rheumatic fever a temporary sinus bradycardia in 25 %.

That the origin of these escaped beats is essentially connected with nervous, especially vagatonically conditioned factors is indicated by the fact that most of the children showed signs of neurolability. The role of such nervous factors is clearly illustrated by the case in which this arrhythmia was only found when the child was lying down, but disappeared when it rose from that position and the pulse frequency increased. This fact that the heart action can increase after changing from a lying to a standing position has been noticed by LINCOLN (1928), JANZEN (1938) and others.

The origin of sino-auricular blocks seems also to be essentially connected with functional nervous factors. This is supported by the occurrence of neurolability in most of the children and the absence of previous infectious diseases in all the cases. The importance of the vagus in the origin of the block is clear from the case in which the arrhythmia disappeared directly after an injection of bellafolin.

In this connection it may be mentioned that none of the children with escaped beats or with sino-auricular block showed a gallop rhythm in the phonocardiogram. Such disturbances are, according to CARLGREN's phonocardiographic investigations, frequently a sign of myocardial damage.

The WENCKEBACH arrhythmia seems to be the relatively most usual of the atrio-ventricular block of the second degree in children (4 of the 5 cases). Of the etiological factors in this type rheumatic infection plays an important part. Besides the ordinary occurrence of other signs of »myocardial damage» in the electrocardiogram, the fact that the block disappeared when the children recovered from their rheumatic infection also favours the idea of an organic basis for the arrhythmia. On the other hand in one case no direct causative factor for the block could be detected. Yet perhaps it is possible that in this case the block was congenital. This opinion is to some extent confirmed by the child's relative youth (5 ½ years) and the fact that the block remained unchanged for an observation period of 9 months. Considering the organically conditioned murmur there is the possibility that a septum defect is responsible for the partial blocking.

In judging the cases of complete atrio-ventricular block it seems as if all 9 cases fulfil the criteria of congenital block. Thus in no case was there a history of previous infectious diseases and in 7 of them bradycardia had been diagnosed before the fifth year of life. Further the normal sedimentation rate is partly in contradiction to the assumption that the block has arisen on the basis of infection. In establishing more in detail the origin mechanism of the block in seems that the phonocardiographic investigations give valuable information. It is remarkable that in all the cases an organically conditioned murmur of high frequency was registered, and its character rather indicated a septum defect. It is thus probable that such an anomaly is the reason for the interruption in the conduction system. A further fact that favours the idea that such a permanent conduction obstacle has an anatomical basis is that the block remained unchanged for an observation period up to 6 years. It is a striking fact that none of the children suffered any inconvenience from the block and that in all the cases the prognosis was good so far as at present can be judged. In this respect the observations differ from the prevalent conception of the prognosis in congenital block in children (NIELSEN 1935). The favourable results in the above cases can probably be mainly attributed to the fact that there was no form of serious heart injury.

To summarise the above: *Among the arrhythmias due to conduction disturbances in childhood often purely functional and organically conditioned forms can be distinguished. To the former group the sino-auricular disturbances in the form of escaped beats or partial sino-auricular block usually belong. Such arrhythmias are generally transitory phenomena without any clinical importance. In contrast to these the partial atrioventricular block is most frequently the expression of a myocardial damage with an infectious basis. Of the two types of block the* WENCKEBACH *arrhythmias seem to be relatively the commonest. The complete atrio-ventricular blocks are in young people often congenital. Phonocardiographic investigations indicate that in such cases a septal defect is a usual cause of the interruption in the conduction. The prognosis is most frequently good both in partial and complete atrioventricular block.*

Rare arrhythmias

The division of the arrhythmias into groups as reported above corresponds to their most usual forms. Besides these, however, there are disturbances in the heart action which, though rare and less important clinically, are of no little theoretical interest. To these belongs the so-called interference dissociation, a term introduced by MOBITZ in 1923. Its characteristic is that besides the sinus node a secondary or tertiary centre gives out rhythmical impulses resulting in contractions which interfere with the sinus beats. This »competition» between two centres sometimes causes complicated electrocardiographical pictures. In contrast to the extrasystoles in which the distance between the heterotopic beat and the preceding normal beat in the individual case is constant, this coupling varies in the pararrhythmic dissociation from beat to beat. The condition necessary for this arrhythmia is that the stimulus formation in the ectopic centre — which under normal conditions is subdued by the impulses from the sinus node — has increased abnormally. As a rule the secondary centre is situated in TAWARA's node, which next to the sinus node works with the highest frequency within the specific muscular system.

In rare cases different pacemakers work completely independent of each other, without any »competition» arising. The necessary condition for this is a protective block which prevents the impulses from the sinus node reaching the ectopic centre, as, for example in complete auriculo-ventricular block.

More details as to the mode of origin of the interference dissociation are still unknown. Among the pathogenetic factors some

authors include the formation of a temporary exsudate within a localised area of the heart muscle (OETTINGER 1935).

The interference dissociation is a rare occurrence, especially in childhood. Thus BAIN 1939 reckons only 5 published cases comprising all age-groups. The youngest person was an 11-year-old boy. According to SHERF and BOYD the disorder is mostly found in connection with digitalis treatment. NÁDRAI 1941 states that he has not seen interference dissociation in infants, but on the other hand he has seen it in later childhood in association with infectious diseases such as rheumatic fever and scarlatina. Among 150 children with scarlatina he found this form of arrhythmia in 5 cases. WICKSTRÖM, however, in his thorough investigations of scarlatina, does not mention a single case. One case has been published by v. KISS (1942) of a 4-year-old boy with a complicated form of interference dissociation which arose in association with scarlatina. In this case the heart action was directed from the sinus node and from two ectopic centres, the one in the auricle the other in the ventricle. The boy recovered.

RISOTTO, NATIN and DA RIN (1938) have observed interference dissociation in connection with diphtheria and they did not attribute to it any prognostic importance.

Among other rare arrhythmias are some disturbances in the heart action of purely vagal origin. One such case has been published by LIND (see below).

In my material 2 cases of arrhythmia were found which can be referred to the groups mentioned above. The one was a par-arrhythmia with interference dissociation, the other, described by LIND (1944), was a complicated arrhythmia of vagal origin.

Case nr. 1 was a boy aged 7 years 2 months. Measles at the age of 6 but no other previous infectious diseases. Normal development. Physician was visited because of dyspnoea from exertion.

12. 9. 1942. General condition satisfactory. No decompensation symptoms. No signs of neurolability. Blood pressure 105/70 mm Hg. The electrocardiogram showed a dominant rhythm of sinus type of 90 beats per minute here and there broken by nodal beats. Besides that there appeared some ventricular beats of left type with varying coupling. P—Q interval 0.13 second, QRS complexes without notches and of right type (electrical

axis + 100°). S—T segments and T-waves showed nothing remarkable. The phonocardiogram taken at the same time showed a physiological systolic murmur with a frequency up to 250 cycles per second.

17. 4. 1943. Exactly the same form of arrhythmia as before.

5. 5. 1943. The arrhythmia mentioned above has now disappeared. No pathological signs in the electrocardiogram. Moderate sinus arrhythmia of 65—90 beats per minute.

14. 2. 1944, 21. 2. 1945 and 31. 1. 1946. The child has developed normally. No disturbances in the apparatus of circulation. Heart action normal.

Case nr. 2 was a boy of 15. Nothing in the case history to show organic heart disease. During the previous year at times a feeling of weight above the heart region and dyspnoea with heavy work. At a routine examination the doctor diagnosed irregular heart action and sent the patient to hospital. When admitted his general condition was satisfactory. Pronounced symptoms of neurolability. Otherwise a very irregular heart action was the only positive physical finding. Blood pressure 110/80 mm Hg.

The electrocardiogram showed, when the patient was in a lying position, a sinus rhythm of 110—120 beats per minute, broken here and there by sino-auricular block accompanied by intraventricular conduction disturbances. P—Q interval 0.18 second. QRS complexes of normal shape with regard to the normal beats (Electrical axis + 65°). S—T segments and T-waves showed nothing remarkable. When examined phonocardiographically a systolic murmur was registered with a frequency of 50—250 cycles per second.

When the patient was examined in an erect position both the sino-auricular and the intra-ventricular block had disappeared. Normal sinus rhythm of 130—140 beats per minute. P—Q interval 0.14 second.

An increase of the vagal tonus caused by gynergen and pilocarpin gave rise to extremely complicated forms of arrhythmia characterised by disturbances in both the stimulus formation and conduction (multiple extrasxstoles, different types of block and escaped beats). With a decreased vagal tonus — in other words, a predominance of sympaticus — brought about by the inhalation of amylnitrite and the injection of eumydrin, the rhythm normalised. Some hours after the administration of these drugs the original arrhythmia reappeared. When the patient was discharged from the hospital the heart action was normal and he remained symptom-free for an observation period of four years.

Discussion. Knowing the influence of the vegetative nerve system on the heart it is often difficult to draw a distinct limit between organically and functionally conditioned disturbances in the heart action. In the first case reported above there was nothing in the case history to justify the assumption that the

arrhythmia was the expression of an organic heart disease. The absence of other clinical symptoms and the relatively transitory nature of the arrhythmia rather support the idea that nervous factors were important etiologically. Perhaps in this case the interference dissociation can be interpreted as a result of the inhibiting effect of the vagus on the sinus node, which in this way loses its control over the stimulus formation in the ectopic centre.

The vagus conditioned origin of the arrhythmia in the second case is shown by the fact that it was present only when the patient was in a lying position and by the effect of the different drugs. LIND's description of this case gives full details.

According to one conception — held by NÁDRAI (1941) and others — complicated arrhythmias such as interference dissociation are rarely found in children because the disturbances are usually extremely transitory. To discover them it is generally necessary to take a series of electrocardiograms during a long period of observation. It is therefore probable that transitory arrhythmias of different kinds would have been found more often under such test conditions. Transitory anomalies such as inverted P-waves were observed in my material in about 10 cases but there was no question there of an arrhythmia.

Summary: Complicated arrhythmias, such as interference dissociation, are seldom found in children. They are usually transitory and have little clinical importance. Their origin seems not infrequently to be associated with nervous factors.

Heart action in premature infants

Different authors have been interested in the question as to whether premature infants show disturbances in the heart action to any special extent. The first more detailed comparative investigations of this subject were made by BURGHARD and WUNNERLICH in 1927. Their material comprised 32 normal new-born infants and a similar number of premature infants of the same age. The observations showed that new-born children often show irregularities in the heart action. Thus 10 cases of extrasystoles were observed in the former group. In the premature infants there were also disturbances, but no real difference could be traced between the children of the two groups. On the basis of these results they concluded that there is no ground for assuming a special »premature ECG».

LONDE (1932) examined 25 premature infants, all under 24 hours old, electrocardiographically and followed their development during the next weeks. In 2 children extrasystoles were registered but disappeared in a few weeks. Such transitory arrhythmias are, as already mentioned, not unusual in fully developed normal children during the new-born period (SMITH 1922, GARRAHAN and LARGUIA 1939, LYON and RAUH 1939 and others).

RÄIHÄ undertook in 1936 a thorough investigation of the heart action in premature children. Among 45 such children he observed no less than 17 with disturbances in the heart action during the first weeks of life. In 10 cases there were extrasystoles. Changes in the electrocardiogram, such as notches in the QRS complexes and

negative P- and T-waves were not rare. Among the etiological factors concerned with the origin of such disturbances in premature infants RÄIHÄ discusses the possibility that they are an expression of a temporary lack of oxygen in the capillary system of the heart. He also thinks that the disturbances can be released reflexively or in consequence of changes in the position of the heart. His records were relatively long, corresponding to an observation period of up to 10 minutes.

Later the heart action in premature children was studied by ENGEL (1937), MIGLIORI (1938) and by NÁDRAI (1938, 1941). ENGEL's comparative investigations were of 8 premature, 44 new-born fully-developed and 75 infants under 1 year. In the premature children extrasystoles were registered in 1 case and in the new-born children in 2 cases. A comparison of the electrocardiograms showed that the curves of the premature children were not more undifferentiated than those of the others. MIGLIORI also came to the same result in a comparative electrocardiographic examination of 21 premature and 23 fully-developed children in the new-born period. NÁDRAI's material is the most extensive and comprises 50 premature, 100 new-born children and 250 infants under 1 year. These investigations also revealed no differences in principle regarding the heart action in children of the different groups. In general the deflexions in the electrocardiograms of the premature children were somewhat less than in the other children, and in addition a relative predominance of the negative deflexions could be observed in the former.

Disturbances in the heart action at an early stage of life can sometimes be traced back to the foetal life. Thus it is known that it is not unusual that the obstetrician diagnoses irregularities in the heart action already in utero. Such arrhythmias, if they are pronounced, usually indicate a threat of foetal asphyxia (HOLTERMANN 1928, 1929). Yet the disturbances can have their cause in the foetal heart itself. According to HEDBERG 1944 they are termed genuine foetal arrhythmias and, in connection with a description of 2 of his own observation, he mentions 20 cases of such arrhythmias as already published. About one-third of these children had different kinds of congenital heart disease. Of the varied forms of arrhythmia

which have been verified after birth complete and incomplete block as well as extrasystoles were the most usual. Recently SØNDERGAARD (1942) examined foetuses electrocardiographically in utero during the last month of pregnancy and this method of registration increases our possibilities of more closely judging the heart action in an early period of life.

The material as the Heart Station included 63 premature infants under 2 months old, of whom only 10 were more than 1 month. There were 31 boys and 32 girls. In all the children both the birth weight and the weight when examined was less than 2500 grams. The following are the main results:

Heart frequency averaged 160 $\pm$ 3.7 beats per minute. Extreme figures were 125 and 200 beats per minute (σ = 28.2).

The figures for the P—Q interval were in all cases between 0.08 and 0.12 second (Average 0.10 $\pm$ 0.002 second, σ = 0.017). The electrical axis of the QRS complexes was between +65° and +165° (σ = 24.1). Average was 109° $\pm$ 3.1°. The breadth of the QRS complex was in 1 case 0.06 second, in all the others 0.03 to 0.05 second (Average 0.04 $\pm$ 0.001 second). In all the cases the height of the complex in lead I was less than 1 millivolt.

The T-waves were positive in 52 children in all the leads. In 9 cases isoelectric and in 2 cases negative T-waves were observed but only in lead III.

Pathological changes in the electrocardiogram were seen in only a few cases. Small notchings in the QRS-complexes were observed in 3 children and in 1 case there were slightly elevated S—T segments in leads II and III. Extrasystoles of supraventricular origin were registered in 1 child (see p. 36). No other certain arrhythmias than physiological sinus arrhythmia were shown in the electrocardiograms. In 2 children (Figs. 14 and 15) in leads II and III some extra waves resembling extra T-waves were observed in a few places, but as they were quite irregular and temporary and did not affect the dominant rhythm they were interpreted as artefacts. In 1 case extra waves resembling auricular fibrillation were observed in the

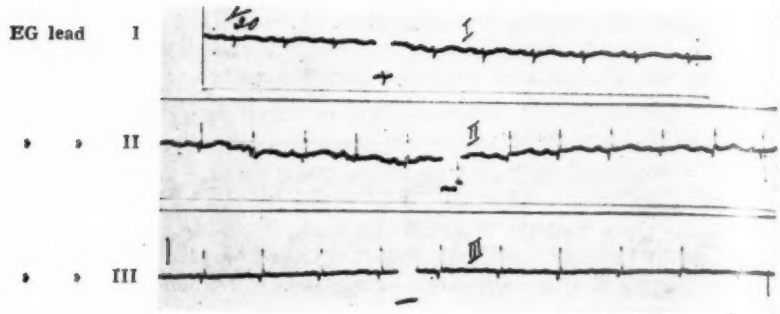


Fig. 14. Electrocardiogram of a premature infant 9 days old. In leads II and III between the QRS complexes some extra waves here and there.

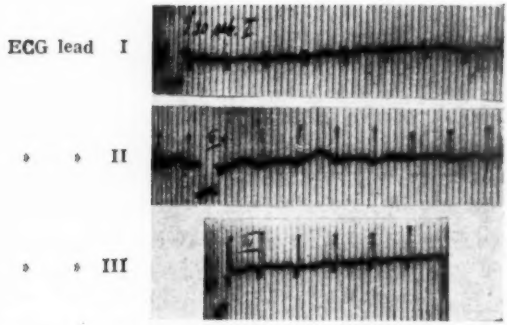
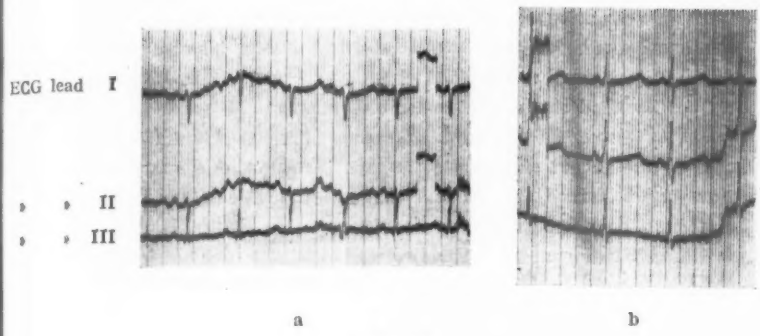


Fig. 15. Electrocardiogram of a premature infant of 1 month. In leads II and III between the QRS complexes extra waves here and there.



a b

Fig. 16.

a. Electrocardiogram of a premature infant 3 weeks old. Several extra waves between the QRS complexes in all leads.

b. Nine weeks later. Normal electrocardiogram.

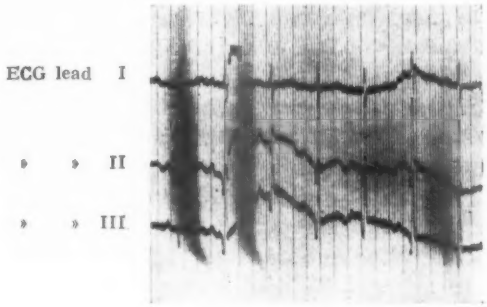


Fig. 17. Electrocardiogram of a premature infant 2 months old. Inverted P-waves in leads II and III in two places.

electrocardiogram in all leads (Fig. 16). These formations, however, were not seen on all sections of the film and as the dominant rhythm itself was regular they also were interpreted as artefacts. The child developed normally and when examined 9 weeks later there were no pathological signs in the electrocardiogram. Finally in 1 child transitory inverted P-waves were seen in leads II and III — a sign of multifocal stimulus formation — but there was no question of an arrhythmia (Fig. 17). None of the children showed any sign of conduction disturbances.

Phonocardiographic examinations were made of 48 of the children. Only in 1 case was an organically conditioned murmur registered with a frequency up to 400—500 cycles per second. The same child also showed the notchings in the QRS complexes mentioned above.

Discussion. If the above results are compared with the observations by different authors of fully-developed, new-born infants it seems as if the heart action in premature children is often distinguished by a relative tachycardia during the earliest period of life. The average heart frequency of the premature infants in my investigations was 160 ± 3.7 beats per minute. HECHT (1913), KRUMBHAAR and JENKS (1917), SEHAM (1924) and NÁDRAI (1941) report corresponding frequences for fully-developed, new-born infants as 121, 124, 139 and 125 beats per minute respectively. In premature infants — the number is not given — NÁDRAI observed an average frequency of 136 beats per minute.

Apart from the relative tachycardia the observations do not seem to reveal in the electrocardiogram any special characteristics in the premature children. The values both for P-Q intervals and for the duration of QRS complexes agree with those given by RÄIHÄ (1936), ENGEL (1937) and NÁDRAI (1941). Neither of the latter authors could show any difference regarding these figures in premature and fully-developed, new-born children.

As regards the position of the electrical axis in premature infants RÄIHÄ gives examples which show that the angle α is less than in fully-developed, new-born infants. Thus he measured a minimum value for it of $+70°$. On the other hand JUNDELL and STENSTRÖM (1931—1932) sometimes found values which are

higher than in fully-developed, new-born children. In new-born children in general the position of the electrical axis of the heart is, according to a summary by MANNHEIMER (1940), ca + 130°. My figures obtained above of 109° ± 3.1° thus seem to indicate a relatively left position of the heart in new-born premature children. Considering the rather great standard deviation ($\sigma = 24.1$), however, no final judgement can be given in this respect.

The rare occurrence of pathological changes in the electrocardiogram is remarkable. Small notchings in the QRS complexes and changes in the S—T segments in at least two leads occurred in 3 and 1 cases respectively. Here it may be stated that NÁDRAI (1941) and PÜTZ and ULLRICH (1942) assert that 20—30 % of all children show transitory signs of »myocardial damage» during the new-born period, chiefly in the form of such changes in the electrocardiogram as have just been mentioned. Compared with the observations reported above this figure seems rather high.

In 1 case transitory negative P-waves were observed which, however, is not quite unusual in children otherwise (see p. 86).

Definite forms of arrhythmia occurred in only 1 premature infant (extrasystoles). Some extra deflexions in the electrocardiogram which were observed in some cases as a transitory phenomenon were accepted as artefacts. This interpretation seems justified, besides for the reason already mentioned, against the background of observations made by NÁDRAI (1941) and CAMMANN (1944) according to which disturbing currents arising from muscular action can give rise to changes in the electrocardiogram resembling disturbances in the heart action, such as auricular fibrillation. These authors specially warn against a misinterpretation of such artefacts. As a matter of fact it is difficult to get an electrocardiogram free from disturbances when dealing with children, so that some authors recommend that they should rather be examined while asleep (SEHAM 1924).

In connection with the method (p. 13) it was stated that the usual length of the films corresponded to a period of 20—30 seconds. It is very possible that irregularities in the heart action would have been observed more often in the premature children if the periods of observation had been longer. This would, however, not

exclude the possibility that fully-developed, new-born infants could also show such transitory disturbances under similar test conditions.

Summary: The examination of electrocardiograms of 63 premature infants of whom 53 were less than 1 month old, has shown that these children did not reveal disturbances in the heart action more often than other children.

Summary.

The investigations here reported were carried out at the Crown Princess Lovisa's Children's Hospital in Stockholm in the years 1945—1946.

Sinus arrhythmia is generally considered as a physiological phenomenon in childhood. Opinions, however, differ as to what pathological importance can be attributed to a higher or lower degree of it. In order to elucidate this question sinus arrhythmia was studied under normal and pathological circumstances. SCHLOMKA's frequency index, RI, was used to determine the degree of arrhythmia. The normal material was composed of 200 healthy children divided into age groups of 0—1, 1—5, 6—10, and 11—15 years. The investigations showed that the degree of arrhythmia was lowest in the age-group 0—1 year and increased successively with increasing age. The pathological material comprised 50 children with congenital heart disease and a similar number with rheumatic heart affection aged 6—10 years. It appeared that the degree of arrhythmia was considerably lower in these cases than in normal children of the same age. This difference was also statistically confirmed as regards the values in the age-group of the normal material showing the lowest degree of sinus arrhythmia (0—1) year. In other words the observations showed that a pronounced sinus arrhythmia indicates a good heart function while in different heart diseases the reflex mechanism is impeded for some reason and this expresses itself in a regular pulse.

In the next section of the work the purpose was to investigate the frequency of the different pathological arrhythmias, their pathogenesis and their clinical manifestations. At the Heart Sta-

TABLE 15.

Pathological arrhythmias detected in material comprising 5600 children under the age of 16 years.

Arrhythmia		Number of cases	Frequency %
Disturbances in the stimulus formation	Extrasystoles	86	1.53
	Auricular flutter	6	0.11
	Paroxysmal tachycardia..	5	0.09
Conduction disturbances	Escaped beats	6	0.11
	Sino-auricular block	7	0.12
	Partial auriculo-ventricular block	5	0.09
	Complete auriculo-ventricular block.....	9	0.16
	Complicated arrhythmias	2	0.04
	Totals:	126	2.25 %

tion at the Hospital 5600 children aged 0—16 years have been examined during the years 1936—1946. Of these children 8650 electrocardiograms have been taken. Most of the children have at the same time been examined by MANNHEIMER's phonocardiographic method, Altogether pathological arrhythmias were found in 126 children, i.e. 2.25 % of the whole material. Table 15 shows the distribution of these arrhythmias.

A more detailed analysis of the arrhythmias can be summarised in the following main points:

Extrasystoles appear not infrequently in clinically healthy children but are relatively more usual in children with myocardial damage or heart disease. The arrhythmia is often associated with neurolability. It appears not infrequently in connection with different infectious diseases. Ventricular extrasystoles are more common than supraventricular. In the latter type clinical symptoms from the heart are more often seen than in the former. There is, on the other hand, no corresponding difference between the different types of ventricular extrasystoles. Repeated examinations were made of 32 of the patients. In 13 of them the extra-

systoles were diagnosed on only one occasion, while in the others the arrhythmia remained during an observation period varying between 1 month and 4 $^1/_2$ years. All the children developed normally and remained well during the period of observation.

Among the other active heterotopias auricular flutter may be considered as a serious disturbance in the heart action, especially in infants. The material included 2 infants, both of whom died. In them the flutter was congenital. In the other cases the arrhythmia mostly appeared in connection with a rheumatic infection. The infants showed distinct symptoms of decompensation, in the elder children such symptoms were less constant. In half of the cases there was congenital heart disease. In 1 child the flutter temporarily became auricular fibrillation after digitalis medication. No other cases of fibrillation were found.

Like flutter, paroxysmal tachycardia has a special position in infancy as its symptoms are essentially different from the corresponding disorder in older children and adults. As the tachycardia in infants generally gives rise to »Vorhofspropfung» with accompanying decompensation such patients present a uniform clinical picture which was formerly misinterpreted, but is now explained, chiefly thanks to American investigators. At times it seems difficult to find any direct etiological factors in the origin of this disorder. Thus the paroxysmal tachycardia appeared in none of my cases in connection with any infectious disease, nor did any of the children show signs of heart disease. The prognosis as to the health was generally good.

Among the arrhythmias which originated in consequence of conduction disturbances escaped beats and sino-auricular block are mostly transitory phenomena without any clinical importance. They usually occur in connection with neurolability in children otherwise healthy. In contrast to these arrhythmias the partial auriculo-ventricular block is not infrequently an expression of myocardial damage often with a rheumatic basis. Of the two block types the WENCKEBACH arrhythmias seem to be relatively the most usual.

Complete auriculo-ventricular block is often congenital. Thus in each of the 9 cases there was reason to assume such an etiology.

When the mechanism of the origin of the block was more closely investigated it appeared that all these children showed a high frequency, organically conditioned murmur whose character chiefly indicated a septal defect. One circumstance giving further support to the idea that the block arose in consequence of such a permanent interruption in the conduction is that the block remained unchanged during an observation period up to 6 years. Both in partial and complete atrio-ventricular block the prognosis is generally good.

Complicated arrhythmias such as interference dissociation are rare, transitory disturbances without any clinical importance. They occur not infrequently in neurolabile children.

Special study was devoted to the heart action in premature infants. The material comprised 63 such infants most of them under the age of 2 weeks. The examinations revealed, with the exception of a relative tachycardia, no special features in the electrocardiograms, and the disturbances in the heart action were not more usual than in other children.

Literature

Amberg, S., and Willius, F. A.: Auricular flutter with congenital heart disease. Amer. J. Dis. Shildr. *32:* 99: 1926.

Antell, L.: Premature Contractions of the Heart in Children. Arch. Pediatr. *48:* 640: 1931.

Arana, R. M., and Kreutzer, R.: Cit. Zbl. Kinderhk. *34:* 368: 1937.

Ask-Upmark, E.: Der Einfluss des Prostigmins auf das normale Electrocardiogramm. Z. Kreisl. forsch. *33:* 41: 1941.

Bain, C. W. C.: Interference Dissociation. Lancet *1:* 20: 1939.

Bainbridge, F. A.: The Regulation between Respiration and the Puls-Rate. J. Physiol. *54:* 192: 1920.

Bass, M. H.: Significance of Cardiac Extrasystoles in Childhood. J. amer. med. Assoc. *86:* 387: 1926.

Beer, A.: Vorhofflattern nach Diphterie. Jb. Kinderhk. *149:* 207: 1937.

—»— Reizleitungsstörungen bei Scharlachfällen die durch verschiedene Gelenkprozesse kompliziert waren. Jb. Kinderhk. *151:* 73: 1938.

v. Bernut, F., and v. den Steinen, R.: Paroxysmale Tachycardie ein Symptom von Encephalitis. Z. Kinderhk. *48:* 686: 1930.

Blumberger, K. J.: Störungen der Reizbildung und Reizleitung des Herzens bei einer zweimaligen Scharlacherkrankung. Klin. Wschr. *2:* 1166: 1936.

—»— Die Unterscheidung zweier Typen von sinoaurikulärem Block. Dtsch. med. Wschr. *62:* 1377: 1936.

Blumenfeld, E. and Putzig, H.: Experimentelle elektrokardiographische Studien über die Wirkung der Respiration auf die Herztätigkeit. Pflügers Arch. *155:* 443: 1914.

Boas, E. P., and Levy, H.: Extrasystoles of Clinical Significance. Amer. Heart J. *11:* 264: 1936.

Bolt, W.: Vorhofflattern bei toxischer Diphterie. Z. Kreisl. forsch. *32:* 345: 1940.

Bower, H. J.: Complete heart-block in young people. Report of two Cases. Lancet *2:* 686: 1939.

Burghard, E., and Wunnerlich, A.: Das Elektrokardiogramm des Säuglings, des Neugeborenen und des Frühgeborenen. Z. Kinderhk. *45:* 56: 1927.

Cammann, O.: Angeborene Störungen der Erregungsüberleitung zwischen Herzhof und -kammer. Mschr. Kinderhk. *66:* 278: 1936.

—»— Beiträge zur Elektrocardiographie im Kindesalter. Mschr. Kinderhk. *82:* 24: 1940, *93:* 76: 1944.

Cammarella, C.: Contributo alla conoscenza della Tachicardia parrossistica nell'infanzia. Riv. Clin. pediatr. *39:* 489: 1941.

Campbell, M.: Etiology and Significance of Extrasystoles. Guy's Hosp. Rep. *79:* 142: 1929.

Campbell, M., and Elliot, G. A.: Paroxysmal tachycardia; aetiology and prognosis of one hundred Cases. Brit. Heart J. *1:* 123: 1939.

Carlgren, L. E.: Gallop Rhythm in Children etc. Acta paediatr. *33:* Suppl. VI: 1946.

Carr, J. G., and Mc Clure, W. B.: Auricular flutter in a new born infant. Amer. Heart J. *6:* 824: 1931.

Carter, J. B.: The fundamentals of electrocardiographic interpretation. Illinois, U.S.A., 1945.

Christensen, J. F.: Paroxysmal tachykardi hos spaede børn. Nord. Med. *22:* 797: 1944.

Clarke, T. W.: Paroxysmal tachycardia in infancy. Arch. Pdiatr. *52:* 666: 1935.

Comby, J.: Arytmie cardiaque et extrasystoles chez les enfants. Arch. Méd. Enf. *42:* 581: 1939.

Cookson, H.: Auricular fibrillation in children. Lancet *2:* 1139: 1929.

Cowan, J., and Ritchie, W. T.: Diseases of the Heart. London 1922.

Currie, G. M.: A case of congenital complete heart-block. The effect of atropine. Brit. Med. J. *1:* 769: 1940.

O'Donovan, P.: Auricular Flutter after Diphteria in a Child. Lancet *1:* 607: 1929.

Drawe, C. E., Hafkesbring, E. M., and Ashman, R.: Children's Electrocardiograms II. Changes in Childrens Electrocardiograms produced by rheumatic and congenital Heart Disease. Amer. J. Dis. Childr. *53:* 1470: 1937.

Dünzelmann, N. L.: Diagnose und Prognose der paroxysmalen Tachykardie im Kindesalter. Mschr. Kinderhk. *93:* 1: 1944.

Mc Eachern, D., and Baker, B. M.: Auricular fibrillation. Amer. J. med. Sci. *183:* 35: 1932.

Edén, L.: Transitory bradycardia during acute rheumatic carditis in children. Ann. Paediatr. *154:* 48: 1940.

Engel, E.: Das Elektrokardiogramm des gesunden Frühgeborenen, Neugeborenen und Säuglings. Z. Kinderhk. *59:* 359: 1937.

Engelmann, T. W.: Über den Einfluss der Systole auf die motorische Leitung der Herzkammer mit Bemerkungen zur Theorie allorhytmischer Herzstörungen. Arch. ges. Physiol. *62:* 543: 1896.

Farr, L. E., and Wegman, M. E.: Extreme tachycardia in the newborn. Amer. J. med. Sci. *190:* 22: 1935.

Faxén, N.: Paroxysmal tachycardia and bundle branch block in a boy of 11. Acta paediatr. *18:* 491: 1936.

Feer, E.: Lehrbuch der Kinderheilkunde. Jena 1941.

Fleisch, A., and Beckmann, R.: Die raschen Schwankungen der Pulsfrekvenz registriert mit dem Pulszeitschreiber. Z. exper. Med. *80:* 487: 1932.

Frédericq, H.: Les fonctions de nerfs accélerateurs du coeur etc. Arch. internat. Physiol. *13:* 115: 1913.

Friberger, R.: Über Arytmie bei gesunden Kindern. Arch. Kinderhk. *58:* 30: 1912.

Frisell, E.: (In the press).

Gaál, J.: Angeborenes Vorhofflimmern. Ann. pediatr. *163:* 165: 1944.

Garrahan, J. P., and Larguia, A. E.: Cit. Zbl. Kinderhk. *37:* 200: 1939.

Garrey, W. E.: Auricular Fibrillation. Physiol. Rev. *4:* 215: 1924.

Geiger, C. J., and Laurence, E. H.: Prenatal diagnosis of complete congenital heart block. J. amer. med. Assoc. *115:* 2272: 1940.

Gibson, S.: Auricular fibrillation in childhood and adolescence. J. amer. med. Assoc. *117:* 96: 1941.

Gjørup, E.: Angeborener vollständiger atrioventriculärer Herzblock bei einen neunjährigen Knaben. Hosp. Tid. 977: 1933.

Goldbloom A., and Segall, H. N.: Auricular fibrillation in infancy. Amer. J. Dis. Childr. *56:* 587: 1938.

Gorter, E.: Cit. Zbl. Kinderhk. *38:* 656: 1941.

Gupta, J. C.: Electrocardiographic studies in a case of heart-block in rheumatic carditis. Indian J. Pediatr. *4:* 17: 1937.

Hafkesbring, E. M., Drawe, C. E., and Ashman, R.: Children's Electrocardiograms. I. Measurements for one Hundred normal Children. Amer. J. Dis. Childr. *53:* 1457: 1937.

Halbertsma, Tj. and Hartog, H. A. Ph.: Auricular flutter in a young infant with recovery on digitalis therapy. J. Pediatr. *15:* 114: 1939.

Halsey, R. H.: The elucidation of some Arrhytmias of the Heart in Children by Means of the Electrocardiograph. Arch. Pediatr. *34:* 128: 1917.

Hecht, A. F.: Der Mechanismus der Herzaktion im Kindesalter, seine Physiologie und Pathologie. Erg. Inn. Med. *11:* 324: 1913.

Hedberg, G. T.: Über die echte fötale Herzarrhytmie und ihre Prognose. Acta obstetr. scand. *24:* 420: 1944.

—»— Foetal arrhytmia — congenital auricular flutter. Acta obstetr. scand. *25:* 399: 1945.

Hering, E.: Über den Einfluss der Atmung auf den Kreislauf. S. ber. Akad. Wiss. Wien *64:* 333: 1871.

—»— Zur experimentellen Analyse der Unrägelmässigkeiten des Herzschlages. Arch. ges. Physiol. *82:* 1: 1900.

Heubner, D.: Über die Bedeutung von Herzblock bei Neugeborenen. Z. Kreisl. forsch. *30:* 600: 1938.

Heubner, O.: Über Herzarytmie im Kindesalter. Z. Klin. Med. *26:* 193: 1894.

Heymans, C.: Der Mechanismus der Herzarythmie respiratorischer Ursprungs. Erg. Physiol. *28:* 292: 1929.

Hobbs, L. F.: Paroxysmal tachycardia in infancy. Amer. Heart. J. *21:* 804: 1941.

Hoekenga, T.: Complete heart block in an infant associated with multiple congenital cardiac malformations. Amer. J. Dis. Childr. *69:* 231: 1945.

Holtermann, E.: Über intrauterine Arrhytmie (Extrasystolie) in der Schwangerschaft. Zbl. Gynäk. 2743: 1928.

—»— Zur Pathologie der übertragenen Schwangerschaft. Ein Frühsymptom des drohenden intrauterinen Absterbens übertragener Kinder ohne erkennbare Ursache. Zbl. Gynäk. 711: 1929.

Holzmann, M.: Klinische Elektrokardiographie. Zürich 1945.

Howard, P. J.: Paroxysmal tachycardia in an infant the fourth day of life. Recovery with digitalis. J. pediatr. *26:* 273: 1945.

Hubbard, J. P.: Paroxysmal tachycardia and its treatment in young infants. Amer. J. Dis. Childr. *61:* 687: 1941.

D'Irsay, S.: On the Meaning of Extrasystoles. Amer. J. med. Sci. *174:* 96: 1927.

Janzen, R.: Die Änderung des Elektrokardiogramms beim Übergang aus dem Liegen in die aufrechte Körperstellung. Z. exper. Med. *103:* 671: 1938.

Johnson, G. D.: Paroxysmal tachycardia in children. Amer. J. Dis. Childr. *60:* 1137: 1940.

Josephtal, F.: Geheiltes Kammerflattern nach Diphterie. Wien. Arch. inn. Med. *26:* 15: 1934.

Jundell, I., and Stenström, N.: A study of the electrocardiogram in infants of normal conditions and during treatment with cod liver oil and vigantol. Acta paediatr. *12:* 113: 1931—1932.

Jörgensen, J.: Pre-excitation i Barnealdern. Nord. Med. *27:* 1655: 1945.

Keith, J. D., and Brown, A.: Paroxysmal tachycardia. Amer. J. Dis. Childr. *59:* 362: 1940.

v. Kiss, P.: Sino-aurikulärer Block bei einem diphterischen Kinde. Mschr. Kinderhk. *66:* 30: 1936.

—»— Dauernder Herzblock nach Diphterie. Jb. Kinderhk. *151:* 347: 1938.

—»— Zur Ätiologie des »Scharlachherzens». Arch. Kinderhk. *117:* 120: 1939.

v. Kiss, P., and di Keppel, C.: Über die Bedeutung der Elektrokardiographie für die Beurteilung von Diphterikranken. Jb. Kinderhk. *147:* 171: 1936.

v. Kiss, P., and Románvi, J.: Zur Ätiologie des Scharlachherzens. Über extrasystolische Arrhytmie bei Scharlach. Arch. Kinderhk. *115:* 226: 1938.

v. Kiss, P., and Voltay, B.: Neuere Beobachtung über Herzblock bei Kindlicher Pneumonie. Arch. Kinderhk. *123:* 85: 1941.

v. Kiss, P., and Wollek, B.: Herzblock beim Kinde im Anschluss an eine kroupöse Pneumonie. Arch. Kinderhk. 104: 38: 1935.

Klemola, E.: Untersuchungen über das Belastungselektrokardiogramm bei Myokardschädigungen nach akuten Infektionskrankheiten. Duodecim. Ser. B. Tom. XXXII, Fasc. 2. 1942.

Koch, E.: Cit. Weber 1939.

Krumbhaar, E. B., and Jenks, H. H.: Electrocardiographic studies in normal Infants and Children. Heart *6:* 189: 1917.

Kühn, M.: Die angeborene atrioventrikuläre Herzblock. Z. Kreisl. forsch. 34: 304: 1942.

Kühne, K.: Die respiratorische Arrhytmie bei Herzkrankungen im Kindesalter. Mschr. Kinderhk *66:* 7: 1936.

Laurentius, P.: Beitrag zur aktiven heterotopen Reizbildungsstörungen des Herzens im Kindesalter, Mschr. Kinderhk. *77:* 30: 1939.

Lebret, J. D., and Erkelens, A. D. Cit. Zbl. Kinderhk. *40:* 468: 1942.

Leconte, M.: L'extrasystole; Valeur sémiologique et pronostique. Arch. Mal. Coeur *4:* 273: 1911.

Leffkowitz, M.: Über Extrasystolen in Schuhlalter. Klin. Wschr. *10:* 1577: 1931.

Lemann, I. I.: Heart block in the young. Ann. Int. Med. *7:* 779: 1933.

Lewis, T.: Single and successive Extrasystoles. Lancet. *1:* 382: 1909.

—»— Observations upon Flutter and Fibrillation. Part. IV. Impure Flutter; Theory of circus Movement. Heart *7:* 293: 1920.

—»— Clinical Electrocardiography. London 1931.

Leys, D.: Paroxysmal tachycardia in infancy. Arch. Dis. Childh. *101:* 44: 1945.

Leys, D., and Russell, H. B.: A case of persistent auricular fibrillation in a child of six. Lancet 702: 1927.

Lichtenstein, A.: Cirkulationsapparaten hos barn. Nordisk lärobok i pediatrik. Frölich, Th., Lichtenstein, A., and Monrad, S. Köpenhamn. *1:* 314: 1945.

Lincoln, E. M.: Tke hearts of normal children. Amer. J. Dis. Childr. *35:* 398: 1928.

Lind, J.: Om vagusbetingade ekg-förändringar. Nord. Med. *24:* 2181: 1944.

—»— »Pre-excitation» of the ventricular part of the heart and its occurence in children. Acta paediatr. *32:* 153: 1945.

Lindqvist, N.: Paroxysmal takykardi hos nyfödd. Nord. med. *4:* 3556: 1939.

Lommel, F.: Klinische Beobachtungen über Herzarhytmie. Dtsch. Arch. klin. Med. *72:* 465: 1902.

Londe, S.: Some Aspects of Circulation in Premature Infants. Amer. J. Dis. Childr. *44:* 110: 1932.

Lukomski, P.: Elektrokardiographische Beobachtungen bei akutem Rheumatismus. Dtsch. Arch. klin. Med. *174:* 268: 1932.

Lyon, R. A.: Disturbances of rate and rhythm of the heart. Textbook of pediatrics. Edited by Nelson, W. E. Philadelphia and London 828: 1945.

Lyon, R. A., and Rauh, L. W.: Extrasystoles in Children. Amer. J. Dis. Childr. *57:* 278: 1939.

Mackenzie, J.: The Study of the Puls. Edinburg and London 1902.

Mannheimer, E.: Elektrokardiografiens användning inom pediatriken. Nord. med. Tskr. *12:* 2039: 1936.

—•— Calibrated phonocardiography and elektrocardiography. Acta paediatr. *28.* Suppl. 11. 1940.

—•— Calibrated phonocardiography. Amer. Heart J. *21:* 151: 1941.

—•— Kalibrierte Phonokardiographie. Cardiologia *6:* 281: 1942.

—•— Paroxysmal tachycardia in infants. Acta paediatr. *33:* 383: 1946.

Meyer, A. G. Cit. Sherf, D., and Boyd, L. J. 1945.

Mengoli, V.: Cit. Amer. J. Dis. Childr. *54:* 1109: 1937.

Migliori, V.: Cit. Amer. J. Dis. Childr. *57:* 1431: 1939.

Mikulowski, V.: Contribution a l'etude de la bradycardie constante chez l'enfant. Rev. franç. Pédiatr. *12:* 805: 1936.

Mines, G. R. Cit. Sherf, D., and Boyd, L. J. 1945.

Mobitz, W.: Zur Frage der atrioventriculären Automatie. Die Interferenzdissoziation. Dtsch. Arch. klin. Med. *141:* 257: 1923.

Nádrai, A.: Die Elektrokardiographie im Säunglingsalter. Z. Kinderhk. *60:* 285: 1938.

—•— Die respiratiorische Arrhytmie bei Diphterie. Ann. pediatr. *152:* 35: 1938—39.

—•— Die Elektrokardiographie im Säuglings- und Kindesalter. Erg. inn. Med. *60:* 688: 1941.

Neubauer, C.: Diphteric Heart Disorders in Children. Brit. med. J. *2:* 91: 1942.

Nielsen, A. L.: Zwei Fälle von angeborenem Herzblock. Acta paediatr. *17:* 554: 1935.

Nordenfelt, O.: Studien über respiratorische Arrhytmie besonders des Kindesalters. Arch. Kreisl. forsch. *13:* 97: 1943.

Noubécourt, P.: Troubles du rhytme cardiaque chez les enfents. Gaz. Hop. *112:* 641: 1939.

Noubécourt, P., and Pichon, E.: Rheumatisme Cardiaque évolutif avec insuffisance auriculaire gauche chez une fille de dix ans Arch. Méd. Enf. *28:* 493: 1925.

Oettiger, I: Zur pathologischen Physiologie und Klinik des atrioventrikulären Dissoziation. Dtsch. Arch. klin. Med. *178:* 117: 1935.

Orgain, E. S., Martin, J. M., Harriot, I., and Anderson, G.: Electrocardiographic alterations in rheumatic fever in Children. Amer. J. Dis. Childr. *62:* 26: 1941.

Pal, J.: Zur Genese und Therapie der Extrasystolen. Med. Klin. *32:* 84: 1936.

Peel, A. A. F.: Congenital block with atrial and ventricular septal defect. Brit. Heart J. *5:* 11: 1943.

Peiper, A.: Unrägelmässige Herztätigkeit im Kindesalter. Dtsch. med. Wschr. *56:* 1079: 1930.

Perry, C. B.: Electrocardiogram of normal school-children. Arch. Dis. Childh. *6:* 259: 1931.

Poynton, F. J., and Wyllie, W. G.: Auricular flutter in infancy. *211:* 371: 1926.

Puglisi, A.: Cit. Zbl. Kinderhk. *37:* 200: 1939.

Püschkel, E.: Das Krankheitsbild der paroxysmalen Tachycardie beim Säugling. Mschr. Kinderhk. *80:* 375: 1939.

Pütz, Th., and Ullrich, O.: Studien über die Bedeutung der elektrokardiographischen Verlaufsbeobachtung des Neugeborenen. Z. Kinderhk. *63:* 136: 1942.

Resnik, W. H., and Scott, W. J.: Auricular Fibrillation. Amer. J. Dis. Childr. 31: 357: 1926.

Risotto, A., Natin, J., and da Rin, C.: Cit. Z. Kreisl. forsch. *30:* 667: 1938.

Rosenbaum, F. F., Johnston, F. D., and Keller, A. P.: Paroxysmal ventricular tachycardia in childhood, Amer. J. Dis. Childr. *64:* 1030: 1942.

Rothberger, C. J.: Normale und pathologische Physiologie der Rhytmik und Koordination des Herzens. Erg. Physiol. *32:* 427: 1931.

Rubell, I., and Strauss, H.: Fatal paroxysmal ventricular tachycardia in a young child. Amer. J. Dis. Childr. *51:* 633: 1936.

Räihä, C.-E.: Das Elektrokardiogramm des Frühgeborenen. Acta paediatr. *18:* 440: 1936.

v. Saalfelt, E.: Venöser Blutdruck und Herzreflexe pulmonalen Ursprungs. Pflügers Arch. *231:* 724: 1932.

Saltzman, F.: Om den fullständigt oregelbundna hjärtverksamheten eller flimmerarytmin. Finska Läk. sällsl. Hdl. *80:* 295: 1937.

Schlomka, G.: Untersuchungen über die physiologische Unrägelmässigkeit des Herzschlages. Z. Kreisl. forsch. *29:* 510: 1937.

Schlomka, G., and Reindell, H.: Untersuchungen über die physiologische. Unrägelmässigkeit des Herzschlages. Z. Kreisl. forsch. *28:* 473: 1936.

Schlomka, G., and Schölmerich, P.: Untersuchungen über die physiologische Unregelmässigkeit des Herzschlages. Z. Kreisl. forsch. *33:* 442: 1941.

Schmitz, H. W.: Auricular fibrillation in Childhood. Amer. J. Dis. Childr. *44:* 310: 1932.

Schwartz, S. P., and Weiss, M. M.: Auricular fibrillation in children. Amer. J. Dis. Childr. *36:* 22: 1938.

Seckel, H.: Herz- und Kreislaufreflexe bei kindlicher Diphterie. Jb. Kinderhk. *143:* 269: 1934.

Seham, M.: Electrocardiogram in normal children. Amer. J. Dis. Childr. *21:* 247: 1921.

—»— Acquired Diseases of the Heart. Pediatrics. Philadelphia, W. B. Saunders Company. 391: 1924.

Semerau, M.: Die Flimmerarytmie. Erg. inn. Med. *19:* 134: 1921.

Sherman, J., and Schless, R. A.: Auricular flutter in a newborn infant. J. Pediatr. *5:* 802: 1934.

Sherf, D., and Boyd, L. J.: Clinical electrocardiography. London 1945.

Shookhoff, C., and Taran, L. M.: Electrocardiographic studies in infectious diseases. Amer. J. Dis. Childr. *42:* 342: 1931.

Siegel, M. L.: The Cardiac Arrhytmias. Therapeutics of infancy and childhood. Edited by Litchfield, H. R., and Dembo, L. H. Philadelphia 1528: 1945.

Sjöqvist, I.: Ett fall av ante partum konstaterat hjärtblock hos foster simulerande hotande fosterasfyxi. Sv. Läkartidn. 1896: 1942.

Smith, A. L.: Clinical Study of One Hundred Patients with Extrasystoles as seen in office Practice. Ann. Clin. Med. *3:* 385: 1924.

Søndergaard, T.: Fosterelektrokardiografi. Ugeskr. Læg. *104:* 775: 1942.

Taran, L. M., and Jennings, K. G.: Paroxysmal atrioventricular nodal tachycardia in a newborn infant. Amer. J. Dis. Childr. *54:* 557: 1937.

Thomson, J.: Congenital Complete Heart Block. Arch. Dis. Childh. *18:* 190: 1943.

Traube, L.: Ueber periodische Thätigkeitäusserungen des vasomotorischen und hemmungs-Nervencentrums. Zbl. med. Wiss. *56:* 881: 1865.

Wallgren, A., and Winblad, S.: Congenital heart-block. Acta paediatr. *20:* 175: 1938.

Walsh, B. J., and Sprague, H. B.: Paroxysmal tachycardia in a child, treated with acetyl-beta-methylcholine chloride (Mecholyl). Amer. Heart J. *20:* 111: 1940.

Weber, A.: Betrachtungen über die rhytmische Herzkontraktion. Z. Kreisl.forsch. *31:* 186: 1939.

—»— Cit. Nordenfelt, O. 1943.

Wegman, M. E., and Egbert, D. S.: Congenital rhabdomyoma of the heart associated with arrhytmia. J. Pediatr. *6:* 818: 1935.

Wenckebach, K. F., and Winterberg, H.: Die unregelmässige Herztätigkeit. Leipzig 1927.

Vesa, A.: Untersuchungen über die Einwirkung des Adrenalins auf das Elektrokardiogramm. Acta Soc. Medic. fenn. »Duodecim». Tom. B XXVI, 2 : 1939.

White, P. D.: Heart disease. New York 1937.

Wickström, J.: Eine Elektrokardiographische Untersuchung über das Herz beim Scharlach. Acta paediatr. *14:* Suppl. 4: 1933.

—»— Wenckebach-arytmi vid scarlatina. Finska Läk. sällsk. Hdl. *77:* 151: 1935.

Winterberg, H.: Vorhofflimmern. Handbuch d. Physiol. *7:* 663: 1926.

Visco, F.: L'estrasistole nell'infanzia. Pediatria *19:* 369: 1911.

Wissler, H.: Ein Fall von angeborenem totalem Herzblock mit Schmerzen in den Schienbeinen. Ann. pediatr. *156:* 53: 1940.

Wolff, L., Parkinson, J., and White, P. D.: Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Amer. Heart J. *5:* 685: 1930.

Wright, F. H.: Paroxysmal nodal tachycardia in a girl 8 years old treated with mecholyl. Amer. J. Dis. Childr. *56:* 1334: 1938.

Yater, W. M.: Congenital Heart Block. Amer. J. Dis. Childr. *38:* 112: 1929.

Yater, W. M., Leamann, W. G., and Cornell, V. H.: Congenital heart block. J. amer. med. Assoc. *102:* 1660: 1934.

Yater, W. M., Lyon, J. A., and McNabb, P. E.: Congenital heart block. J. amer. med. Assoc. *100:* 1831: 1933.

Öhnell, R.: Pre-excitation a cardiac abnormality. Acta med. scand. Suppl. 152: 1944.

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